

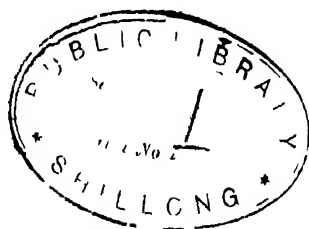
# FUNGI AND DISEASE IN PLANTS

*An introduction to the  
diseases of Field and Plantation Crops, especially  
those of India and the East*

By

E. J. BUTLER MB. FLS

Imperial Mycologist Agricultural Research Institute, Pusa



CALCUTTA and SIMLA  
THACKER, SPINK & CO  
1918

PRINTED BY  
THACKER, SPINK & CO  
CALCUTTA



## PREFACE.

THIS book is the outcome of a request made to me by Mr. James Mollison, C.S.I., when Inspector-General of Agriculture in India, that I should prepare a handbook on the crop diseases of India caused by fungi. At the time the field was almost unexplored, and it will still be long before it can be fully covered. British India is a large country, and there are few areas under a single Government in any part of the world that contain a greater variety of crops, or where individual crops are found growing under a greater variety of climatic and soil conditions. It is not in a few years, nor even in a lifetime, that the diseases of each of these crops in every locality can be examined.

But the book has been much delayed, and the fact that there are still large gaps in our knowledge is no longer a sufficient reason for deferring publication. Nearly 200 diseases of Indian crops are included, of which the great majority have been practically studied in the laboratory and in the field. In the temperate north-west most of the diseases found are already known in Europe or the United States; in the tropical south only a few. The former are easy to identify, and the experience of more advanced countries can be applied to their control, often with but slight modifications. The latter are more difficult, since few tropical countries have as yet progressed far in the study of plant diseases. Apart from this, India is sufficiently isolated to have developed many indigenous diseases, and in their study no outside assistance is available: under sugarcane, for instance, it will be noticed that nearly half the diseases described are unknown elsewhere.

Mr. Mollison's intention was that the book should be primarily for the use of the trained staff of the Agricultural Departments in India. Long experience had convinced him that the damage to crops by pests of various kinds was so great that one of the most promising fields of agricultural activity in India was the study of methods of reducing it. That he was right has been abundantly proved by subsequent experience with such cases as sugarcane diseases and the cereal smuts. The smuts of jowar alone are calculated to cause an annual loss of over a million sterling in the Bombay Presidency, and practically the whole of this is preventable.

A second class for whom an account of Indian crop diseases will be useful is the considerable body of planters, many of whom, though primarily concerned with special crops such as indigo, sugarcane, tea, coffee, and rubber, are also interested in general farming. To these may be added a small but increasing number of Indian landowners who are commencing to develop their estates by more scientific methods of agriculture. In the Colleges which were founded under the policy of agricultural development initiated by Lord Curzon, the number of boys who are studying agriculture with a view to return to the land is increasing, and for these some knowledge of plant diseases beyond what they get at college is necessary. But the practical man is not concerned with the more technical aspects of the subject, and the latter have therefore been usually given in small type in the following pages.

Further, the study of natural science in all its branches is, after a long period of neglect, beginning to take its proper place in the Universities and University Colleges in India. The materials and information dealing with the structure and life of the lower plants have hitherto perforce been drawn from European sources, and it is well that there should be available some account of the commoner fungi actually found in India.

Outside India also it is hoped that a work dealing with plant pathology may serve at least to call attention to the enormous preventable waste of resources caused by vegetable parasites, especially in the tropics. The study of plant diseases has been much neglected by us as a nation in the past—the recent disastrous shortage of potatoes in the British Isles proves how serious may be the result of such neglect—but there are signs of an awakening, and it is certain that we cannot afford, in these times of stress, to lag behind most other countries of the civilized world in this matter.

The present volume deals with field and plantation crops only. Fruit and forest trees are omitted, and few garden crops are referred to. Their inclusion would have made the volume unwieldy, and would have been difficult on account of the absence of information regarding the diseases of several of our most important trees. The study of these is being taken up as opportunity permits, and it will doubtless be found necessary at some future time to deal with them in a second volume.

Pusa,  
March, 1918. }

E. J. BUTLER.

# CONTENTS.

## Part I.—GENERAL.

### CHAPTER I.

	PAGE.
THE NATURE OF FUNGI .. .. .	3
Limits of the group — The structure of fungi — Mycelium — Sporophores — The reproduction of fungi — Formation of spores — Types of spore — Sexual reproduction — Polymorphism — The dissemination of fungi — Classification.	

### CHAPTER II.

THE FOOD OF FUNGI .. .. .	41
Nature of the food — Saprophytes — Parasites — Mechanism of feeding by parasites — Preparation of the food for absorption — Effects of different kinds of food — Symbiosis.	

### CHAPTER III.

LIFE-HISTORY OF PARASITIC FUNGI .. .. .	58
Persistence from season to season — Heterocism — Specialisation of parasitism — Infection.	

### CHAPTER IV.

THE CAUSATION OF DISEASE BY FUNGI .. .. .	75
Definition of disease — Diagnosis — Symptoms — Morbid anatomy.	

### CHAPTER V.

THE PRINCIPLES OF THE CONTROL OF PLANT DISEASE ..	105
Conditions influencing the severity of disease — Virulence — Predisposition — Immunity — Selection and breeding of resistant varieties — Deterioration — Epidemics — Methods of direct control — Seed disinfection — Soil disinfection — Spray fluids — Powders.	

# CONTENTS.

## Part II.—SPECIAL DISEASES.

### CHAPTER VI.

	Page.
CEREALS .. .. .	151
Diseases of wheat — Oats — Barley — Maize — Jowar — Bajra — Rice — Lesser millets.	

### CHAPTER VII.

PULSE CROPS .. .. .	244
Diseases of pigeon pea — Peas — Beans — Cowpea — Soy bean — Val — Khesari — Lentils — Velvet beans — Gram — Guar.	

### CHAPTER VIII.

VEGETABLES, ROOT-CROPS, AND OIL-SEEDS .. .. .	274
Diseases of Potato — Brassicas and allied plants — Tomato — Brinjal — Bhindi — Kachu — Cassava — Cucurbits — Celery — Chua — Bathua — Kulfa — Groundnut — Linseed — Castor.	

### CHAPTER IX.

DYE, DRUG, AND SPICE CROPS .. .. .	333
Diseases of tobacco — Opium — Turmeric — Ginger — Chilli — Pepper — Coriander — Fennel — Fenugreek.	

### CHAPTER X.

FIBRES .. .. .	363
Diseases of cotton — Jute — Sann hemp — Sisal.	

### CHAPTER XI.

DISEASES OF SUGARCANE .. .. .	377
-------------------------------	-----

### CHAPTER XII.

DISEASES OF TEA .. .. .	413
-------------------------	-----

### CHAPTER XIII.

DISEASES OF COFFEE .. .. .	467
----------------------------	-----

### CHAPTER XIV

DISEASES OF RUBBER .. .. .	490
<i>Acknowledgements</i> .. .. .	517
<i>Bibliography</i> .. .. .	518
<i>Index</i> .. .. .	533

**PART I.**  
**GENERAL.**



## CHAPTER I.

### THE NATURE OF FUNGI.

IN our ordinary speech, the term "plant" conveys at once the idea of the trees, shrubs, and herbs that form the green covering of the earth. The idea is readily extended to the seaweeds and slime-weeds found in water. A step further will take us to the larger fungi—the mushrooms and toadstools—clearly to be included amongst the vagaries of plant life; and a step but little more difficult to the white or coloured mould fungi which appear on decaying fruit or jam, on old wood in damp localities, or on boots or other leather articles in the hot, moist weather of the rainy season in India.

But beyond these limits there is still a vast mass of forms of plant life, often unrecognised as such. These include the bulk of the parasites that cause damage to crops, and so they have an importance as plants altogether out of proportion to their size. In almost any field of grain in India, the blights known as rust and smut can be found. In the one there are small masses of rusty powder on the green parts of the cereal, in the other a sooty matter filling the grain. These are genuine plant structures, but they are not a part of the cereal on which they are found. The little yellow or black masses consist of multitudes of tiny, seed-like bodies (known as "spores," for they are not quite the same as the seeds of higher plants), belonging to a fungus, the rest of whose body is buried within the tissues of the cereal and only to be seen by special methods of examination. The dust-like spores each can germinate and form a little shoot, which grows, puts out branches, again produces spores, and dies. Though invisible without the aid of the microscope, the whole process is much the same as in other, more familiar, plants, and each seed-like spore is just as capable of giving a new fungus plant as any one of our field or garden plants is of growing from its proper seed.

Accepting then this fact that fungi are plants, we must next see what are the limits of the group. What plants are to be considered as fungi? It is impossible to give a simple and complete answer to this question. As we approach the lower confines of the vegetable kingdom,

the boundaries of the different groups become less distinct ; it is not always easy to separate the fungi from the other main groups of lower plants, such as the algæ, the bacteria, and the Myxomycetes or slime-fungi.

The algæ (seaweeds and their allies) are distinguished from the fungi by one mark of great importance. Like the flowering plants, they obtain their carbon food from the carbon-dioxide of the air, by means of a green matter known as chlorophyll, through the energy of sunlight. Chlorophyll is entirely absent from fungi. The effect of this on their nutrition is considerable. The flowering plants, the ferns, mosses, algæ, in fact all the green plants, build up their food out of inorganic salts dissolved in water from the soil, together with the constituents of the air. The fungi are unable to do this, but require as an essential part of their food organic matter, or food already prepared by being built up into the bodies of plants or animals. In most cases they live by feeding on the bodies of plants and animals, either alive or dead, or on such substances as once formed part of these, such as jam or leather. There are other broad distinctions between fungi and algæ, but none that can be relied on to the same extent as the above. For instance, most fungi live on land, most algæ in water ; but intermediate forms are found, as in the Phycomycetes or alga-like fungi, many of which live in water, whereas several algæ live on trees or in moist soil on land. So also the structure of the higher members of the two groups is very different, but many Phycomycetes might pass for algæ except for their lack of chlorophyll.

The bacteria and Myxomycetes are still often included under the fungi, and this fact alone shows how difficult it is to find a satisfactory distinguishing mark between these groups. Like the fungi, they are without chlorophyll and are dependent on other plants or animals for their organic food.

The bacteria are, however, as is well known, extremely minute bodies, usually consisting of a single cell or of a collection of more or less independent units. Sometimes these units are arranged in branching threads, which are hard to distinguish from some of the fungi proper. The fungus thread is, however, normally a part of the one individual, whereas a bacterial thread consists of a chain of individuals more or less loosely joined together. The branching also is different, the fungus having true branches, while in a bacterial column the spurious appearance of branching is due to imperfect separation of broken parts of the chain.



which continues to give off new individuals above and below the break. The most important distinctions between the two groups are in the methods of growth and reproduction. Fungi usually grow only at the ends of the threads which compose their bodies, and multiply by forming spores at the ends of, or within, special threads. Bacteria, on the other hand, have not got this purely end growth, and usually multiply by each cell simply dividing into two cells, which frequently separate from each other and become independent. There are other minor differences such as the presence of true nuclei in fungus cells and their absence in bacteria, the nature of the cell wall, and so on.

The Myxomycetes or slime-fungi approach the fungi on the one hand and the lower members of the animal kingdom on the other. They have been for many years often claimed alike by botanists and zoologists. They have a power of spontaneous movement; from the spore issues a small lump of naked protoplasm which crawls about until it meets with other similar bodies, all then uniting to form a mass of protoplasm, sometimes yards in extent, which is also capable of slow motion. Eventually it becomes stationary and forms spores resembling fungus spores. The structural differences and peculiar life of the Myxomycetes are sufficient to place them in a distinct group, though they are perhaps related to the fungi. A few are plant parasites but only one of these has as yet been found in India.

Another group of plants which may sometimes be mistaken for fungi is the lichens. These are familiar to everyone, encrusting the surface of rocks and the bark of trees. Each really consists of a combination of an alga and a fungus, living together in mutual co-operation, each assisting the other in the all-important matter of obtaining food. The green alga is able to supply organic food manufactured by the aid of its chlorophyll from the air. The fungus protects the alga with a coating of strong threads, and supplies mineral food from the substratum. That the association is benefited by this partnership is shown by the enormous number of these plants, and the great variety of situations in which they are to be found. They are often injurious to trees and shrubs by forming a crust on the bark or leaves, but are not usually true parasites as they do not feed on the living substance of their host.

### The structure of fungi.

Excluding the above groups, the chief structural characters of fungi may next be considered. All known fungi, with very few exceptions,

originate from spores, bodies comparable with, though not strictly similar to, seeds. These spores germinate,

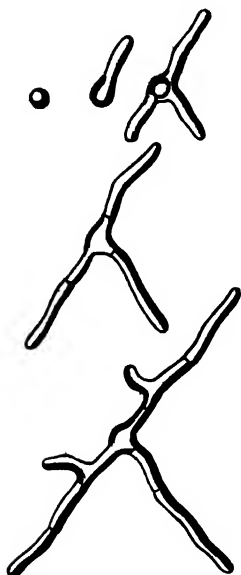


FIG. 1. Germination of the spore of *Penicillium*. (After Zopf.)

shape : in the most primitive group of fungi, the Chytridiaceæ, this type is common.

**STRUCTURE OF THE MYCELIUM.**—At an early period in its growth, the germ-tube and the hyphæ which arise from it usually become segmented into a row of cells, by the formation of transverse walls (septa) across the filament. Each cell is a hollow structure, bounded by walls of a pliable, transparent substance, which is in some cases composed of the chemical compound cellulose, best known as forming the bulk of such vegetable fibres as cotton or flax. In most cases, however, the wall largely consists of a substance closely resembling chitin, the material which forms the hard covering of insects.

Within the walls, the cell is filled with a watery jelly, the protoplasm, a material found in all living bodies and which is, in fact, the actual living matter itself. The protoplasm does not fill the cell all through with a

uniform mass. Here and there little hollows or spaces, called "vacuoles," occur, containing water with dissolved substances, the whole forming the cell sap. Here and there also condensations of a peculiar form of protoplasm form what are known as "nuclei." In segmented filaments, each cell usually contains one nucleus only. In certain cases, such as some stages in the Ascomycoetes, smuts, rusts, and Basidiomycetes proper, two nuclei occur habitually in each cell, and in others three or more

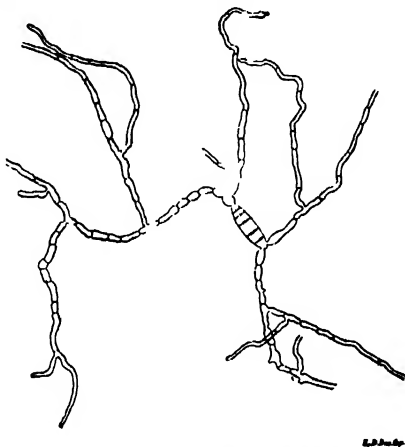


FIG. 2 Mycelium from germinated spore of *Helminthosporium Sacchari*.

have been found. In the lower fungi, the mycelium is ordinarily unsegmented, no transverse septa being formed. In such cases, the nuclei are distributed irregularly in the protoplasm and there is no hint at separate cell formation. The precise functions of the nucleus are not fully known, but it is certain that it is the bearer of the hereditary characters of the cell, and also that it plays an important part in regulating the nutritive changes that occur in cells.

The essential part in the fungus plant, as in other plants, is the protoplasm with its contained nuclei. The cell wall is not essential since it is absent in the swimming spores of lower fungi and in the vegetative body of some Chytridiaceæ. So also, vacuoles are not found in most young growing cells. Numerous other substances occur in fungus cells, none of which are essential. Thus, crystals of calcium oxalate have

been found in a few species, and crystalline albuminous bodies are frequently present in the moulds (*Mucors*). Sugar in solution is

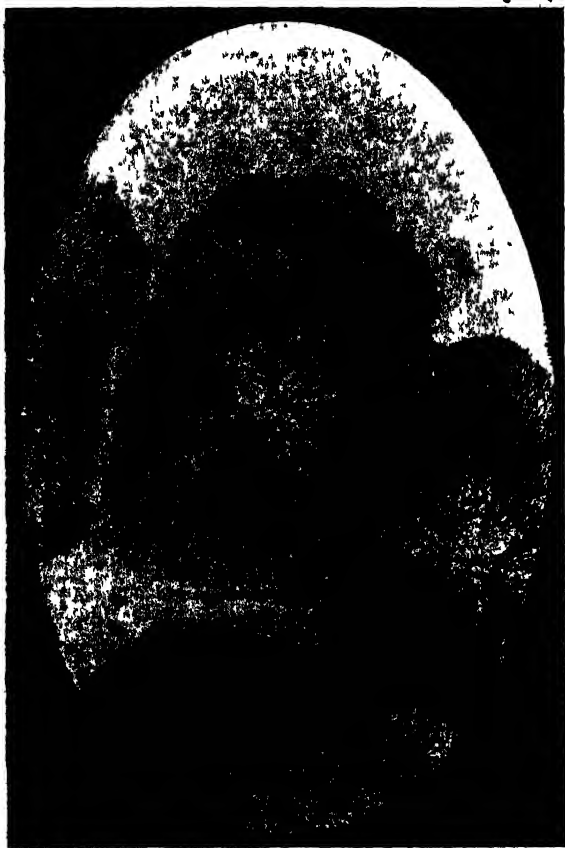


FIG. 2. Mycelium of *Mucor Proteus*, showing part of four plants.

common ; so also is a carbohydrate, glycogen, which largely takes the place of the starch so universal as a food reserve in the higher plants. Fats and oils are abundant in many cells ; pigments also, as one would

expect when recalling the bright colours of many fungi. Resin is found in the cells of some mushrooms. In many fungi, the cell wall is variously modified after formation, the most common change being a gelatinous or gummy degeneration. Infiltration of the membrane with pigments occurs frequently, and with resin and other substances less often.

The body of the simpler fungi usually consists of branching hyphæ only. In many cases, unions termed "anastomoses" occur between neighbouring hyphæ, which either fuse together where they come into contact, or one puts out a short branch whose tip reaches the other and unites with it (Fig. 5, 1-3). In these cases, the cell wall disappears

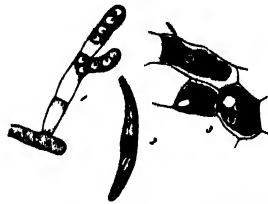


FIG. 4. Fungus cells: 1, *Galactinia saccosa*; multinucleate cell below and binucleate cells above; 2, uninucleate spore of *Vermicularia* sp. from Carica; 3, uninucleate and binucleate cells of the stroma of same. (1 after Maire.)

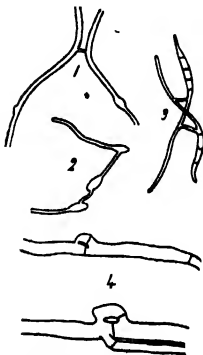


FIG. 5. Anastomoses in 1, *Hendersonina Sacchari*; 2, *Cephalosporium Sacchari* (germ-tubes); 3, *Fusarium udum* (germ-tubes); 4, clamp-connections in *Rhizoctonia*.

of a considerable number of fungi.

The majority of fungi have, however, at least a part of their body, often that part on which the spore-bearing hyphæ are situated, of more complex structure than this. The individual hyphæ unite to form bundles or masses, frequently of characteristic shape. At the same

at the point of contact and the two tubes open into one another. Such unions are common between the germ-tubes of certain fungi. Sometimes two adjacent cells of a hypha unite by a connection around the dividing septum. A little branch grows out just on one side of the septum and curves round until its tip reaches the cell on the other side. Union then takes place and an open passage is formed around the septum. At a later stage this side passage is often again closed by a new septum. These "clamp-connections," as they are termed, are frequently found in the group of higher fungi (Basidiomycetes) to which the mushrooms and their allies belong (Fig. 5, 4). Excluding the formation of spores or reproductive organs, this simple mycelium of branching hyphæ, with or without anastomoses, forms the whole body

time, instead of each cell which is formed during the growth of the hypha retaining the thread-like shape, round or angular cells are commonly produced. The large masses of fungus tissue familiar to everybody as mushrooms consist of immense numbers of hyphæ intertwined in all directions. In some cases, the intertwining is loose and the individual threads can be made out if the mass is separated out by needles in water. In others, hard masses are formed by the fusion of adjoining cell walls, and, as the cells are in these cases frequently rounded or angular, a thin section examined under the microscope shows a tissue of cells closely united together without any trace of thread-like structure. To such a tissue the name "pseudoparenchyma" is often applied, as it resembles the cellular tissues (parenchyma) of certain parts of the higher plants. It is only by following the development of these cell masses that one can ascertain that they are formed by the union of separate hyphæ. In some cases, air spaces are left between the hyphæ, and the tissue is spongy in texture. In others, the whole of the mass consists of cells closely united without any vacant interspaces, and hard, woody or horny structures result. Great modifications of the cell walls often occur in these tissues. Sometimes the wall is much thickened and very hard; such walls are often black or coloured. Sometimes the wall softens into a gelatinous substance, as in many of the fungi known as Tremellinaceæ; when moist these fungi are soft and gelatinous, but become hard, shrunken, and horny when dry. So great is the formation of mucilage from the cell walls of some fungi that it drops from them to the ground.

The simplest form of hyphal mass results from the growth of a number of hyphæ side by side to form an elongated bundle or strand. In most cases, these strands are composed of hyphæ which are all similar, and may be either loosely interwoven without actual fusion or closely united by the lateral adhesion of the wall of adjoining hyphæ. Such strands are common in the soil, particularly where there is much decaying organic matter, as in forests. They belong chiefly to the mushroom group (Basidiomycetes).

Somewhat more complex are the mycelial strands of several fungi allied to the puff-balls. In these, the centre consists of brown, rather gelatinous, thick hyphæ, while on the outside is a layer of thin, white, spirally woven threads, from which short branches project from the surface. Still more highly differentiated strands are found in other Basidiomycetes.

Such specialised strands are known as "rhizomorphs" and they may reach a considerable degree of complexity of structure in some cases.

In the "horse-hair" fungi, found in jungles and tea gardens, smooth, shining, hair-like, black cords run over the aerial parts of the plants on which they grow. The component hyphæ run parallel and adhere to each other, the outer ones being blackish brown and the inner hyaline. In the injurious parasite of the deodar in the Himalaya *Fomes annosus*, tough rhizomorphs somewhat resembling the finer roots of the tree, but much blacker, are found passing from root to root or extending into the soil. They are branched cylindrical or flattened cords, of uneven diameter not diminishing regularly as roots do and consist of an outer black, rather brittle rind and an inner very tough and flexible pith. The rind is composed of brown hard rather thick walled cells united into a pseudoparenchyma in which the original filamentous character of the constituent hyphæ is in great part lost. In the pith, on the other hand the hyphæ persist as fine silky, colourless filaments which can be teased out in water into a bundle of threads. In the common European parasite of tree roots, *Agaricus melleus*

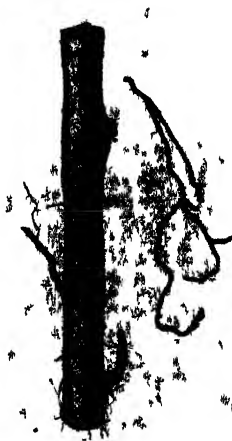


FIG 6 'Horse hair' fungus on *Shorea robusta*. To right, rhizomorph of *Fomes annosus*

(not yet recorded in India) the rhizomorphs are still more complex, as the pith is composed of large elongated cells with thinnish walls united laterally so that the individual threads cannot be separated out.

All these mycelial strands are capable of growth at the tip, sometimes extending for yards and branching and anastomosing freely. Their use is readily understood. Single hyphæ are delicate and easily injured, as by insects. The rhizomorphs are usually tough and hard to damage. By their vigorous growth they permit of extensive spread. They are also, owing to their structure, able to withstand drying or other adverse conditions much better than single hyphæ, and their vitality is such that old, dried rhizomorphs, if brought into a moist atmosphere, can recommence growth and put out new branches even after some years.

Instead of uniting to form long, cylindrical, root-like strands, the hyphæ of some fungi join into large, solid, more or less rounded and sharply defined masses, known as "sclerotia." These masses are very long-lived and resistant to adverse conditions; their cells become filled with stores of reserve food; they usually separate off from the mycelium and become isolated and free; and they can resume growth when conditions become favourable, giving either a new mycelium or in many cases producing the reproductive stage of the fungus.

In some sclerotia, a hard outer part (rind or cortex) may be distinguished from a softer central pith (Fig. 7, 3). The rind is formed of one or more layers of rounded or angular cells, with thick and often coloured walls and small cavities. The pith may also be of pseudo-parenchymatous structure, or the filamentous character of the hyphæ may be preserved. In either case the wall is usually less thickened than in the cortex and is often colourless, while the cell cavities are larger and are rich in reserve food substances. In other sclerotia the structure is fairly uniform throughout. Sometimes hyphæ grow out from the surface of the rind and



FIG. 7. Sclerotia: 1 and 2, *Sclerotium stipitatum* from white-ants nest,  $\times 2$ ; 3, *Rhizoctonia* on jute stem above,  $\times 3$ ; and same from cowpeas in section below,  $\times 330$ .

give a hairy or woolly appearance to the sclerotium; in the older stages these external hyphæ usually wear off. In the development of the sclerotia, two types may be distinguished. In the one, the whole body is the product of a single hypha. This becomes segmented into short cells and gives out lateral branches, which segment in their turn. The branches grow parallel with, and adhere to the sides of the parent hypha and by the constant repetition of this process a mass of solid fungus tissue results. As the segments of each branch are very



short, the distinction between individual threads is soon lost, and the tissue takes on the structure of pseudoparenchyma. In the other type, a number of distinct hyphæ take part in the formation of the sclerotium, all branching freely and giving off a mass of intertwined filaments which, by repeated segmentation and lateral fusion, may lose all trace of their filamentous structure, though the latter is sometimes preserved, especially in the central part of the sclerotium.

Some remarkable instances of sclerotium-formation occur in India. One is common in the nests of white-ants, a rounded or pear-shaped, blackish body of extremely hard texture, often a couple of inches across (Fig. 7, 1—2). It is known as *Sclerotium stipitatum* (Tamil: "Puttunga") and belongs to a fungus which appears on the surface of the nests (*Xylaria nigripes* Kl.). It is commonly believed to have valuable medicinal properties. In the Nilgiris another underground form occurs, *Mylitta lapidescens*, known locally as "little man's bread" in allusion to a tradition that the Nilgiris were once peopled by a race of dwarfs. A similar, but much larger, *Mylitta* occurs in Australia, reaching sometimes to the size of a football. It is known as "black fellow's bread" and, like the Nilgiri species, is eaten by the indigenous tribes. The Australian *Mylitta*, under favourable conditions, resumes growth and forms the fruit body of a large fungus, *Polyporus Mylittæ*, but the further development of the Indian species has not been seen. Another curious form is the "tiger's milk" of the hill tracts of Assam and Malaya. This is a large structure of quite extraordinary hardness, which gives rise to the fruit body of *Polystictus sacer* (Fig. 12, 1). Other sclerotia, usually of small size, are formed by various parasitic fungi. These may occur within the plants attacked, but are more often on the surface. They may be an important means of disseminating the parasite, as they frequently become separated and cast loose in the soil, or can be carried with fragments of disintegrated plant debris, or alone, through the air. In some cases they are the only known means by which the species is propagated, as in *Sclerotium Oryzæ* (p. 230), some *Rhizoctonias* (p. 262), and the sclerotial disease of sugarcane (p. 408).

Besides these resting or durable bodies of considerable size, formed generally by the union of many hyphæ, a number of fungi form resting cells, singly or in small groups. These are found even in the simple filamentous fungi. Portions of a hypha become divided into rounded or angular cells, with a much thickened and usually coloured wall. In these, reserve food is accumulated. Such resting cells may be separated from the hyphæ on which they are borne, and after a period of rest,

or if placed in favourable conditions of moisture and the like, can put out fresh hyphæ, behaving just like a spore. To such vegetative organs of propagation the name "chlamydospore" is often applied. Scarcely

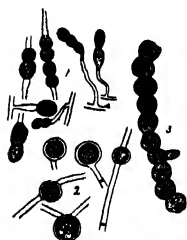


FIG. 8 Chlamydospores :  
1, *Fusarium udum*,  $\times$   
330; 2, *Phytophthora*  
*Colocasiae*,  $\times$  160; 3,  
*Sclerotium Oryzae*,  $\times$   
230.

to be separated from the chlamydospores are the cells usually termed "gemmae," which differ only in that they are not thick-walled, durable cells and that they frequently continue to bud out similar cells to themselves instead of giving new hyphæ. These chlamydospores and gemmae are found even amongst the Phycomycetes or lower fungi, in which, as already mentioned, the hyphæ are ordinarily unsegmented. Under certain conditions of nutrition the mycelium may be almost wholly transformed into masses of rounded cells. Indeed the familiar group of fungi known as yeasts (Saccharomycetes) may be considered as an extreme case of the reduction of the mycelium

to minute gemmae, endowed with a remarkable faculty for budding out similar cells, so as to form colonies which readily break up into their

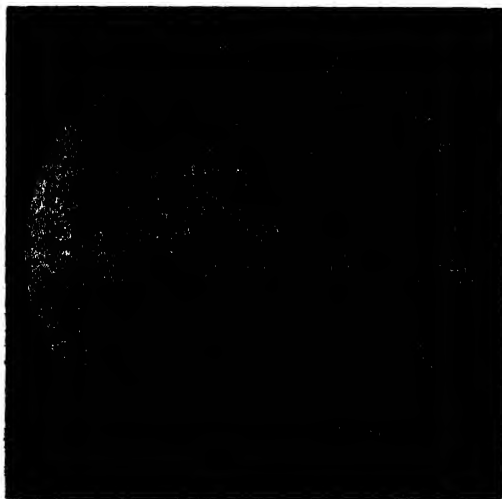


FIG. 9. Gemmate mycelium of *Mucor Pratinii*.

individual units. In a number of cases the formation of gemmæ is associated with fermentative activity, and gemmate fungi—yeasts, *Mucors* and the like—are largely used in brewing.

Another form of specialised mycelial development remains to be considered. In many fungi, after a period of vegetative growth in the

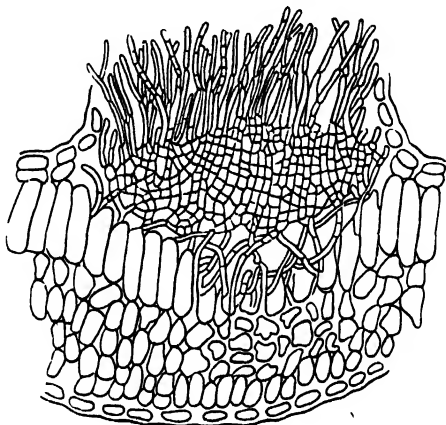


FIG 10 Stroma of *Septoglaum Mori*.

filamentous condition, the mycelium condenses here and there into pseudoparenchymatous masses. These are found generally on or just below the surface of the substratum in which the fungus is growing, and differ from sclerotia only in having usually a less regular shape and a margin less sharply delimited from the rest of the mycelium, the connection with the hyphæ below being maintained. They often, indeed, only form flattened crusts covering the deeper hyphæ from which they are produced, though in other cases they are thick and hardy, if at all, to be separated from sclerotia. These masses are called "stromata." In very many instances, they form a transitional stage between the purely vegetative and the reproductive parts of the fungus body, the spore-bearing organs being developed on their surface or immersed in them, and the term "stroma" is often restricted to such cases. It is difficult, however, to accept this restriction, as it is not uncommon to find stromatic crusts or masses in no way associated

with the formation of spores and even in fungi not known to produce spores, as some species of *Rhizoctonia*.

The parts of the fungus plant so far considered are chiefly concerned with nutrition. The more specialised rhizomorphs, sclerotia, resting and budding cells, take part also in propagating the species, but this is "vegetative" propagation, on a par with propagation by cuttings, buds, suckers, and the like amongst higher plants. The reproductive system proper is usually quite distinct and well defined.

**SPOROPHORES.**—As the vegetative system is usually buried in the substance on which the fungus feeds—in the soil, in rotting organic matter, or in living plants—and as it is necessary that the spores should be formed in free access to the air so as to secure their dissemination, it is evident that some special arrangement must be made to bring the spore-forming parts where they will be exposed to the elements. This is done usually by the development of structures known as "sporophores," raised vertically above the level of the general mycelium. The sporophore may consist either of a single hypha (simple sporophore) or of bundles or complex masses of hyphæ (compound sporophore), and it bears on some part, usually towards the top, the spores of the fungus. The hyphæ of which it is composed ordinarily differ from those of the vegetative mycelium in one or more characters, such as their vertical position, limited growth, or special structure (nature of cell wall, shape of the cells, characteristic mode of branching and the like), and as they may be arranged in systems of great complexity, an almost endless series of different types of sporophore exists.

The simplest forms are found commonly in many of the lower fungi and in those moulds that are familiar on damp or rotting organic substances, and that belong to several different families of the fungi. In some of these, particularly such as have a considerable development of aerial mycelium in addition to the hyphæ buried in the substratum, and some of those that grow in water or in soil, special sporophores are almost or quite dispensed with, as being unnecessary to secure free dissemination. The spores are formed at the tip of, or laterally on, ordinary hyphæ or, in the aquatic species, in special cells on the hyphæ, and are liberated in the air, the water, or the soil as the case may be (Fig. 11, 1—2). More often, however, specialised sporophores are formed, and in the simplest cases these are single hyphæ, often of distinct shape and size, which grow out into the air and bear the spores on the tips or sides or on lateral branches (Fig. 11, 3).

In other cases, clumps of hyphae growing close together rise into the air. Sometimes these merely remain near together without lateral union (Fig. 11, 4), sometimes they unite more or less closely into solid columns (Fig. 11, 5).

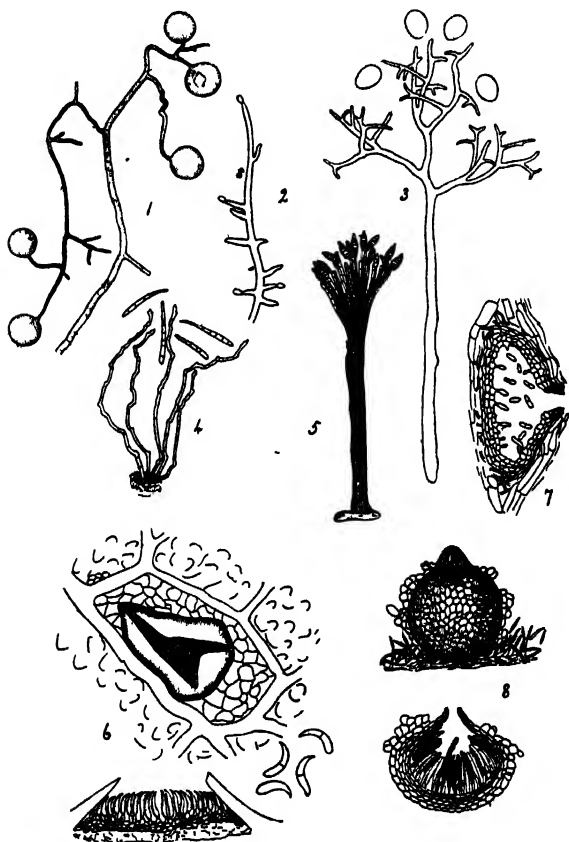


FIG. 11. Sporophores: 1, *Pythium de Baryanum*,  $\times 330$ ; 2, *Cephalosporium Sacchari*,  $\times 350$ ; 3, *Peronospora Viola*,  $\times 210$ ; 4, *Oenosporea longipes*,  $\times 130$ ; 5, *Arthroretium* sp.,  $\times 35$ ; 6, *Marasmius Juglandis* in surface view and section; 7, *Ascochyta Pisi*; 8, *Neotria Holboellii* in surface view and section.

Sporophores aggregated into crusts or layers are produced in many fungi. Sometimes these are formed on the surface of the substratum in which the vegetative mycelium grows, sometimes, especially in parasitic and other fungi that grow on the stems and leaves of plants, just below the surface, which is eventually ruptured so as to expose the spore-bed (Fig. 11, 6). In either case, the layer of sporophores may arise directly from the mycelium below or there may first be formed a stromatic



FIG. 12. Sporophores. 1, *Polystictus sacer* showing both surfaces of expanded cap, and stalk arising from a sclerotium,  $\times \frac{1}{2}$ ; 2, *Schizophyllum commune*,  $\times \frac{1}{2}$ .

tissue composed wholly or in part of pseudoparenchyma. Common examples of spore-beds formed below the surface of the plant are the "sori" of the cereal and other rusts, and the "aecervuli" of the anthracnoses. When formed on the surface, there may be a very large mass of stromatic tissue intervening between the vegetative mycelium and the layer of sporophores. Such compound sporophores in the form of superficial stromatic crusts or tuberoles may be found commonly on pieces of old wood, dead branches of trees, and the like, in damp situations. Sometimes

the edge of the crust becomes free and curves over to form a sort of bracket. This is the first stage in a process which has as its purpose the lifting of the spore-bearing part of the sporophore above the object on which it is growing. In more advanced types, so much of the crust may leave the surface that only a narrow band or stalk remains to join it to the latter (Fig. 12, 2). In this way the bracket fungi found on trees and old wood, and the laterally attached toadstools, are formed. Finally, the whole spore-bearing part of the fructification may be raised from the surface on a central stalk (Fig. 12, 1) and may be expanded into a cap at the top, as in the well-known mushrooms, or may remain as a simple or branched, vertically, elongated body of varying shape.

In another type of compound sporophore, the spore-bearing hyphæ are enclosed in roundish or cup-shaped or flask-shaped receptacles. To a common form of this type the name "pycnidium" is applied when it bears spores on the outside of the fertile hyphæ (Fig. 11, 7), and "perithecium" when the spores are enclosed in the sac-like hyphæ (asci) characteristic of the group of fungi known as the Ascomycetes (Fig. 11, 8). The round sporophores of the puff-balls also belong to this group and there are other examples. These receptacles may be formed either on the surface of the substratum or immersed in it, in the latter case usually opening to the surface by a narrow neck through which the spores are expelled when ripe. They serve the purpose of protecting the spores from injury during their period of development. Sometimes they are formed singly, sometimes in groups immersed in a common stroma, and, as in the last type, the stroma has in some cases become vertically elongated so as to raise the spore receptacles above the level of the substratum.

In most compound sporophores, spores are not produced on all parts of the organ. In the mushrooms, for instance, they are formed only on the surface of the gills found on the under side of the expanded "cap." To the fertile part of the sporophore, i.e., that part which actually bears spores, the name "hymenium" is given.

In many cases, where the spore-bearing stalks are so numerous as to form definite layers or a hymenium, certain sterile hyphæ, usually of characteristic form, occur intermingled with the fertile ones. These are frequently known as "paraphyses" and are especially common in the Ascomycetes and rusts (Fig. 13, 2—3).

From the above account of the fungus body, it will be seen that an ordinary fungus consists of a vegetative part, usually formed of thread-

like hyphæ buried in the substratum, and of a reproductive part which is sometimes not distinct from the vegetative except in bearing spores, but is more usually modified to form a simple or compound sporophore. As the production of spores provides for a new generation of the fungus, we may consider that the life-history of any species begins with the germination of the spore and ends with the development of new ripe spores, or, in the case of those fungi which have more than one kind of spore, with the development of the final or, as it is often termed, "perfect" spore form.

### The reproduction of fungi.

The reproduction of fungi, that is to say the production of a new generation from the old, is secured by the cutting off of specialised cells from the fertile hyphæ, which develop into spores. When ripe, these spores separate from the mother plant and are capable, on germination, of giving rise to new individuals. In certain cases, the spore is formed after the contents of two separate cells fuse wholly or in part. This is termed sexual reproduction. In very many cases, however, the spores are formed asexually, without any preceding fusion of two cells.

**FORMATION OF SPORES.**—The spores of fungi present a wonderful range of variation, there being hundreds of types differing in size, shape, structure, and mode of formation. The one fungus may have several different kinds of spore during the course of its complete life-history. In the vast majority of cases they are very definitely characterised bodies, differing sharply from the vegetative cells of the mycelium. According to their mode of development, and leaving aside for the present the sexually-produced forms, two main types may be distinguished.

1. The spores are formed by the dividing up of the inner contents of a mother-cell into a number of cells, which become separated from one another, as a rule, while still enclosed within the mother-cell, and are ultimately set free by the rupture of the wall of the latter. This is called "endogenous" or "free-cell" spore formation.

2. The spores appear as protrusions of the tip or at the side of the fertile hypha, and are cut off, when they reach maturity, by a septum which usually breaks through and sets them free when ripe. This is called "exogenous" spore formation.

*Endogenous formation.* The first type is common in the lower fungi (Phycomycetes), including most of those that live in water, and also in the



large group known as the Ascomycetes. In the former case the spore mother-cell is termed a "sporangium," in the latter an "ascus."

In the Phycomycetes, the sporangium is usually formed at the end of a hypha, which in most cases swells up into a round, oval, or cylindrical cell. Less often it is formed in the course of a hypha ("intercalary" formation). The contents are rich in protoplasm, often with one or more vacuoles and crowded with nuclei. Round each nucleus a portion of

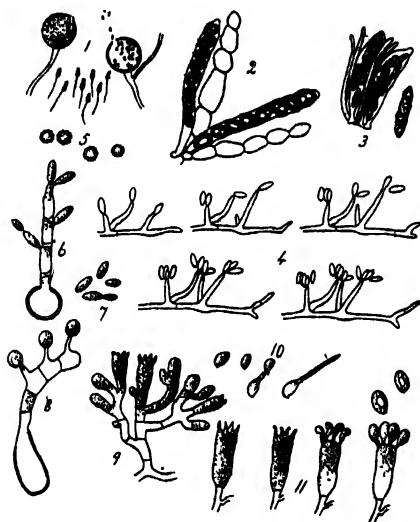


FIG. 13. Formation of spores: 1, sporangia and ascospores of *Plectidium Cuculus*,  $\times 220$  and  $450$  resp.; 2, asci, ascospores, and paraphyses of *Neocosmospora vasinfecta*,  $\times 220$ ; 3, asci and ascospores of *Leptosphaeria Saubhari*,  $\times 250$ , the single spore,  $\times 470$ ; 4, successive stages in formation of conidia of *Neocosmospora vasinfecta*,  $\times 250$ ; 5, spores of *Ustilago Zea*; 6, promycelium and sporidia of *Melanospicium austro-americanum*; 7, sporidia of same; 8, promycelium and sporidia of *Kushnola Fiet*; 9, basidia of *Hypocynus Solani* showing sterigmata; 10, basidiospores of same (two germinating); 11, successive stages in formation of basidiospores of *Polyporus hispidus*, with two mature spores.

the protoplasm collects, and spaces appear between these portions. Finally, as many little spores are formed as there are nuclei. The mother-cell then opens and the spores are released. In the aquatic species, the spores are capable of swimming by the movements of one or more

oar-like threads, the "cilia." Such spores are known as "zoospores," or animal-like spores, from the fact that they are capable of independent movement. They are preserved in several allied forms which occur on land, as in many of the downy mildews which attack plants. To ensure germination in these cases, it is necessary that the sporangia (which when ripe easily fall off from the sporophores) should be immersed in water. As they are very small, the films caused by dew on the leaves, or small drops of rain, are quite enough for the purpose. After lying for a short time in water, the tip of the sporangium opens and the spores escape and swim off by means of their cilia (Fig. 13, 1). When they emerge, they have no wall but consist of naked protoplasm. After a while they come to rest, form a cell wall around them, and germinate by putting out a germ-tube which, if the spore happens to be in contact with a suitable plant, can penetrate the leaf and cause a new patch of disease. In some sporangia the number of spores is very great, as many as 7,000 having been estimated to occur in a single sporangium of an aquatic fungus, *Pleolpidium inflatum*; in others, only 3 or 4 may be formed.

Another type of sporangium occurs in the common moulds known as *Mucors*, land fungi which grow on decaying fruit, bread, and the like. In these the spores are incapable of independent movement. When liberated by the disintegration of the sporangium wall, the minute spores are blown about as dust. It is well known how quickly food and other organic substances become mouldy if left exposed, especially in damp situations. This is due to the presence of spores of *Mucors* and other fungi in the air, from which they are deposited as dust and germinate in the presence of a suitable food supply.

In the *Ascomycetes*, the asci are formed singly at the ends of the fertile hyphæ, usually many crowded together within, or on the surface of, the compound sporophore (Fig. 13, 2—3). In the great majority of cases each ascus contains 8 ascospores. Sometimes there are fewer, sometimes more, in the latter case often a higher power of 2, as 16, 32, 64, and 128, but sometimes an irregular number. This is brought about by successive divisions of the typical eight nuclei of the ascus or by suppression of one or more spores. Occasionally the spores originally formed become septate, and each segment breaks off as an independent spore; in other cases the first-formed spores give rise to others by budding.

The spores formed in sporangia proper are all unicellular, each consisting of a single cell with one nucleus. In very many asci, however, multicellular spores are formed. When first developed these also are

unicellular, but, as they ripen, septa are formed, the nucleus first dividing, so that two or more cells result. In this case, each cell is often able to give out a germ-tube when germination takes place.

*Exogenous formation.* Spores produced in the second main mode, by exogenous spore formation, occur as the only type in the fungi known as Basidiomycetes (including the rusts and most of the large fungi such as mushrooms) and are found as a secondary spore form in many Ascomycetes and some Phycomycetes. In the majority of cases, they are formed by the cutting off of a protrusion from the top or side of the fertile hypha. The portion cut off is the spore, and on ripening it usually falls off. The spores of this type are ordinarily disseminated by the wind.

In the simplest case, the spore is merely the (usually swollen) end of the fertile hypha, or of a small stalk from the end or side of the latter (Fig. 13, 4). The swelling is cut off by a septum from the parent stalk. Very often, instead of a single spore being thus formed, a chain of spores arises from the sporophore. These chains may be developed in either of two ways. Most commonly, each new spore of the chain is successively cut off from the tip of the parent hypha, so that the end spore of the chain is the oldest ("basipetal" formation). Less often, the first-formed spore buds out another at its tip, and this in its turn another, so that the end spore here is always the youngest ("acropetal" formation). As there may be more than one bud from the tip of a spore, the chains of the second type are frequently branched.

After a spore or chain of spores is formed on the end of the sporophore, the latter may continue to grow from a point just below the tip. The new growth usually tends to preserve the same direction as the main stalk, so that the spore is pushed over laterally. This process may be repeated several times.

Instead of the fertile hypha giving off a single spore or chain of spores, it often branches at the top, and each branch produces spores in a similar fashion at its end. Or, instead of branching, the end of the fertile hypha may enlarge into a head which bears a number of spores or of chains of spores, and similar heads may also be borne laterally on the sporophore lower down.

A special form of exogenous spore formation has become the characteristic type in the large group of allied fungi which constitute the family of the Basidiomycetes (Fig. 13, 6—11). In these, the top of the fertile hypha, or of lateral branches from it, forms a special fertile cell, usually

of definite shape. From this cell a limited number (typically 4) of small, slender stalks grow out, each of which bears a spore at its tip. The fertile cell is termed a "basidium," and it may either remain undivided or divide into a limited number of cells (again usually 4) by transverse or vertical walls, each cell giving out ordinarily a single spore-bearing stalk. The stalks are called "sterigmata" and the spores "basidiospores" or "sporidia," the latter name being applied to the basidiospores of the smuts and rusts, in which the basidium is often known by the name of the "promycelium." The term basidium is sometimes loosely applied to any simple sporophore of limited growth, and shape differing from that of the normal hyphæ, especially if formed in a fertile layer, as in pycnidia, and if short and unicellular; in the same way, any short stalks arising from the tips of fertile hyphæ and bearing spores or chains of spores, are often called sterigmata. It seems better, however, to restrict the use of these terms to the Basidiomycetes.

**TYPES OF SPORE.**—According to the manner in which they are formed, to structure, or to other peculiarities, different names are applied to different types of spores. As already mentioned, the swimming spores of aquatic fungi and their allies are termed zoospores. The spores formed within the asci of Ascomycetes are often called ascospores. These of the Basidiomycetes, basidiospores or sporidia.

In species where, besides a formation of spores in sporangia or asci or on basidia or promyelia, a second type of spore, produced exogenously, occurs, this second spore form is usually termed a "conidium." Thus many Ascomycetes possess conidia as well as ascospores, though both may not be simultaneously formed. Similarly, the Basidiomycetes sometimes possess conidia, usually formed on a simple mycelium, in addition to their basidiospores or sporidia; the Phycomycetes also, though less commonly than the other groups. In many fungi ("Deuteromycetes" or "imperfect fungi") a conidial form of spore is alone known, though a number of these cases have been found on closer study to be merely conidial stages of Ascomycetes or Basidiomycetes. Conidia formed in enclosed receptacles (pycnidia) are often called "pycnospores" or "stylospores." In such fungi as the rusts, where a number of spore forms are known in one and the same individual, special names may be applied to each. Thus a perfect rust fungus may possess spermatia, aecidiospores, uredospores, teliospores, and sporidia, all differing in shape, structure, and mode of formation, and most with different properties or uses.

**SEXUAL REPRODUCTION.**—What is termed sexual reproduction in fungi is the union of two more or less specialised cells to produce a spore or a brood of spores, which, when ripe, germinate and give origin to one or more new individuals. The spore form which arises as a result of this union is often termed the “perfect” stage of the fungus. Sexual reproduction is most regularly developed and can be most easily observed in the water-inhabiting fungi and their allies (Phycomycetes). In a few of the most primitive of these, two apparently similar zoospores unite to form a sexual spore, which germinates and gives a new plant. In the more advanced forms, certain cells of the mycelium become enlarged, rounded, and filled with dense protoplasm, usually containing many nuclei. All the last generally degenerate but one—the female nucleus. The whole cell is known as an oogonium. Certain other cells develop from the mycelium, come into contact with the oogonial wall, and put out one or more outgrowths which penetrate the oogonium and allow a nucleus—the male nucleus—to pass into it, usually accompanied by a certain amount of protoplasm. These male cells are termed antheridia. The male and female nuclei fuse together to form a single nucleus, and this, with a great part of the protoplasm of the oogonium, becomes surrounded by a wall to form a single-celled perfect spore, lying within the oogonium. This spore is known as an oospore. It germinates, usually after a period of rest, giving rise to a number of zoospores in some cases, or in others to a single or several germ-tubes.

In the moulds known as Mucors, the two cells that unite are often similar in shape and size, so that it is not possible to distinguish a male and a female cell. In some of these, fusion only takes place between cells arising from different mycelia, that is to say, from hyphæ which have come from different spores. In others, the fusing cells may have originated from one and the same spore. The cell which results from this fusion is termed a zygospore and is capable of giving rise to a new individual on germination.

In some of the Ascomycetes, a type of sexual reproduction similar in its early stages to that of the aquatic fungi occurs. Instead, however, of the two nuclei uniting in the oogonium to form a single spore, the oospore, each divides several times and a male and female pair pass into a group of cells which grow out from the oogonium. From one or more of these cells the asci arise, each ascus still having a male and female nucleus. Fusion of the two nuclei occurs in the young ascus and the ascospores arise by the division of this sexual nucleus. There are various

modifications of this process, in some cases there being no functional male cells, and the fusing nuclei being both female. It is also held by some that the two nuclei do actually fuse in the oogonium, the fusion in the ascus being a second one of unknown significance.

In other Ascomycetes, a peculiar type of sexual reproduction occurs, which bears a resemblance to the type found in some algæ. The male cells are formed from a special cell and become separated off and set free as small independent bodies. One or more of these settle on a second special, elongated cell, the "trichogyne," into which, it is said, the contents pass and travel down until they enter the female cell. Fusion of the male and female nuclei then occurs as before, the fertilised female cell giving rise to asci. It seems doubtful, however, whether the contents of the male cell actually reach the female cell; in some cases at least, the trichogyne is not functional and the male cells degenerate.

In the smuts, a reduced type of sexual reproduction occurs, in which two uninucleate cells fuse to form a binucleate cell. This may take place early in the life-history of the species, as in oat smut, where the germ-tube (promycelium) which is formed when the perfect spore germinates, is composed of 1-nucleate cells. Any two of these cells may unite by a small branch which grows round from one to another, so that a single 2-nucleate cell is formed. The hyphæ which arise from this cell are composed of 2-nucleate cells and the 2-nuclear condition persists through the whole subsequent growth of the fungus up to the formation of its spores. During the ripening of the spore the two nuclei fuse, so that the mature spore has only a single nucleus. Often, instead of fusion occurring between two cells of the promycelium, two 1-nucleate sporidia fuse, the nucleus of one migrating to the other, which then germinates and gives a 2-nucleate mycelium as before. The latter method is the regular one in bunt of wheat, and is found combined with the other in oat smut and several other cases. In maize smut, on the other hand, these early fusions do not occur and the mycelium is 1-nucleate up to the commencement of spore-formation. Then the ends of the hyphæ segment into short 1-nucleate lengths, and the dividing wall between each pair dissolves, leaving a larger 2-nucleate cell. This becomes the spore, the two nuclei fusing as the spore ripens. Hence the perfect spore form of the smuts arises as a result of the fusion of two nuclei, but the sexual act, by which these two nuclei were brought into the one cell, may have taken place far back in the life of the fungus.

The rusts follow an essentially similar method. Here the change from the 1-nucleate to the 2-nucleate condition takes place just prior to the formation of aëdiospores, and is caused by the passage of a nucleus from one cell of the stroma found at the base of the compound sporophore (the aëdium) to a neighbouring cell. The resulting 2-nucleate cell grows out into a chain of 2-nucleate aëdiospores. The 2-nucleate condition persists in the germ-tube of the aëdiospores and throughout the whole of the next spore stage (the uredo stage) and it continues up to the formation of the perfect spore form, the teleutospore. In the teleutospore the two nuclei at last fuse. The germ-tube of the teleutospore (the promycelium) is composed of 1-nucleate cells, the sporidia borne on it are also 1-nucleate, and the mycelium resulting from the germination of the sporidia remains 1-nucleate until the aëdiospores are again about to form. Hence the teleutospores of the rusts are comparable to the perfect spores of the smuts in being the product of a sexual act, though this usually has occurred far back in the life of the fungus.

In the Basidiomycetes proper, the few cases so far observed show a still more reduced type of sexual reproduction than that of the smuts and rusts. The sporidia which arise from the basidium have each a single nucleus and the mycelium which arises from the germination of the sporidium is composed at first of 1- or 2-nucleate cells; later on, as the cells which lead up to the basidia are formed, they are usually regularly 2-nucleate. It is not yet clear how this 2-nucleate condition comes about. There is evidence, however, that the clamp-connections between neighbouring cells of a hypha serve to effect the transfer of nuclei from one cell to the other. The clamp forms in a 2-nucleate cell just above a wall which divides the cell into two 1-nucleate portions. Each of the two nuclei divides and one from the upper pair passes through the clamp to reach the lower cell. Each subsequently formed cell remains 2-nucleate until the basidium develops. In the young basidium the two nuclei fuse into a single "sexual" nucleus and the process is terminated. Hence the only cell union is between two succeeding cells of the same hypha, though it does not seem to be clear as yet whether the two nuclei thus brought together are closely related or not.

### Polymorphism.

One of the most curious circumstances in the life-history of fungi is their capacity for assuming different forms at different periods of their full development. To this the name "polymorphism" is given.

Many interesting cases are known and their number is constantly being added to. Owing, however, to the small size of fungus spores, the similarity of the vegetative mycelium of many species, and the difficulty of being sure that any particular mass of mycelium has arisen from only one and not two or three different spores, it is necessary to exercise great caution in regarding different forms as belonging to the same species. Mere occurrence close together, or the appearance of several types of spore on what is apparently the one mycelium, is not sufficient. It is necessary to make sure by repeated observation and by excluding all sources of error that the one form grows out of the other and that the two are connected together at some stage of their development.

It is, however, quite possible in the laboratory to separate out a single spore and cause it to germinate. The mycelium from this single spore can then be grown in closed vessels, absolutely secure from danger of the entry of any foreign spore. If when thus grown, the fungus gradually assumes different forms or produces different types of spore, one may feel satisfied that all belong to the same plant.

Sometimes, instead of two spore forms appearing successively, both may occur together, or there may be overlapping, the second appearing while the first still persists. In these cases, it is often possible to trace a direct hyphal connection between the two spore forms under the microscope. Again, the spore of one type may on germination give a second type of spore after a longer or shorter interval of mycelial growth; this may happen so quickly that it is easy to see the connection between them.

The Ascomycetes and rusts are particularly rich in polymorphic species, as are also the "imperfect fungi," which are often merely stages in the development of Ascomycetes or Basidiomycetes. Polymorphism, however, occurs to some extent in all groups, though it must not be confused with the formation of sexual spores of distinct type, which is rather to be considered as the completion of development. Polymorphism is confined to the asexual stages of the life-history; we never find two types of spore resulting from the sexual act in any fungus; and in every group of fungi (omitting the Deuteromycetes as having an incomplete life-history) there is a single definite type of spore, the "perfect" spore, which is the result, immediate or delayed, of a sexual act. Many polymorphic forms will be found included in the descriptions of particular parasites in later pages, but one or two examples may be given here.



A species of rust known as *Uromyces Faba* is common on peas, broad beans, and lentils in some parts of India. The first sign of its appearance is an outbreak of tiny, yellowish dots in clusters on the stem and leaves. On examining these under the microscope, they are found to be flask-shaped sporophores, sunk in the tissues and containing a large number of minute spores formed on the tips of short stalks lining the interior of the flask. This kind of spore receptacle of the rusts is called a "spermagonium" and the spores produced within it are known as "spermatia," as they are believed by some to be male cells which have become functionless. Very soon, larger, yellow eruptions, the "æcidia," appear in the same parts of the stem and leaves as the first, and on the same mycelium. These are cup-shaped bodies, the bottom of the cup being formed of a basal stroma and the sides of a lateral membrane, known as the "peridium," composed of a layer of pale, angular cells. This membrane projects above the surface of the leaf or stem and when mature forms a serrated rim to the cup. Within the latter, chains of rather large, yellow "æcidiospores" arise from the basal stroma. A little later, pale brown pustules, the uredo sori, appear scattered all over the plant. These consist of flattened beds of fertile hyphæ, producing single spores the "uredospores" at their tips. Still later, large, blackish-brown pustules are found, chiefly on the stem. These are the teleuto sori, in structure similar to the last, but bearing the perfect type of spore, the "teleutospores," at the tips of numerous stalks arising from the base of the sorus. On germination, the teleutospore produces a single short germ-tube (promycelium) divided by transverse septa into 4 cells, from each of which a sterigma grows out and bears a single small "sporidium" on its tip. Each of these five forms of spore differs from the others in size, structure, colour, or some

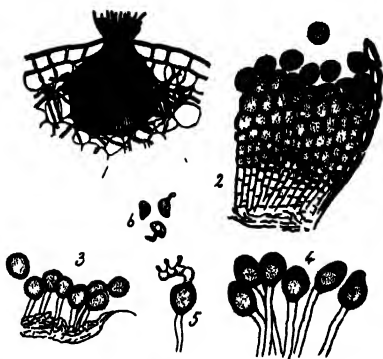


FIG. 14. *Uromyces Faba* : 1, spermagonium,  $\times 100$  ; 2, part of an æcidial cup,  $\times 215$  ; 3, part of uredo sorus,  $\times 215$  ; 4, teleutospores,  $\times 215$  ; 5, germination of teleutospore ; 6, sporidia, two germinating,  $\times 200$ . (1 and 6 after de Bary, 5 after Plowright.)

other particulars. By sowing the sporidia on the host plant, a mycelium bearing spermatogonia and aecidia is produced; the aecidiospores are set free into the air and germinate on other parts of the plant, giving rise to a mycelium which bears uredo sori; when the uredo spores germinate they may give further uredo sori, or teleuto sori, or sometimes both together on the same mycelium; and the teleutospores finally germinate (usually the following year) and give sporidia again.

The "sooty moulds" which are very common on mango, fig, and other trees all over India, belong to another group of highly polymorphic fungi, being Ascomycetes extraordinarily rich in conidial forms. Coffee bushes attacked by bug in south India are often almost completely blackened by the growth of one of these species, *Capnodium brasiliense*, which lives in the sugary "honey dew" produced by the insect on the surface of the leaves and twigs (Fig. 197). As in the rusts, each conidial form in this group has received a separate name, the connection between the different forms not having been suspected when they were first observed. In the species on coffee, the simplest type of reproductive body results from the breaking up of certain hyphæ into chains of nearly cubical cells, which separate readily from one another and are capable of germination. To this form, the name *Torula* is applied. It is probable that its spores belong rather to the chlamydospore type of propagation than to the true conidial type, and the same may also be true of the next form. This consists of compact collections of roundish cells, formed by the division of a cell of the mycelium in all three dimensions. The result is a little packet of cells which remain united, though the whole mass may separate from the mycelium before germination occurs. This spore-type is known as *Coniothecium*. On other hyphæ, short stalks grow out laterally and each bears an elongated, narrow conidium of considerable size and characteristic shape, at its tip. This is the *Brachysporium* type. Similar lateral stalks may bear another kind of conidium composed of three or four radiating arms united at the centre. Each arm consists of a row of four or five cells, the whole forming a multicellular conidium of very peculiar shape. In some sooty moulds, such as that found on coffee in Ceylon, only this form, which is known as *Triposporium*, has been observed. The next type of fructification belonging to *Capnodium brasiliense* is a pycnidial form, which is sometimes, but not invariably, found on affected bushes. The pycnidia are long, cylindrical bodies, sometimes swollen in the middle and occasionally forked. They contain large numbers of very small pycnosporangia, which are liberated through a mouth at the top. Finally, there is a perfect form bearing

ascospores, which seems to occur infrequently. It consists of somewhat irregularly shaped perithecia, containing asci, in each of which are 4 to 8 ascospores. Any one of these spore forms can reproduce the fungus, in all its various shapes, on germination.

It often happens in polymorphic fungi, and in those that have an asexual, as well as a sexual mode of reproduction, that one or other of the forms appears but rarely, or that whereas in one country several forms occur, in another some of these may never be found. Thus the spermatogonia and æcidia of the black rust of wheat have never been found in Australia, though the other forms are common. In India also they are absent, except in the Himalaya, though millions of wheat plants bear the fungus in its other stages every year in many parts of the country. The Ascomycetes have many species which are amongst our commonest moulds in the conidial stage and only rarely produce ascospores; in this group, too, the powdery mildews (*Erysiphaceæ*) are often found in the conidial form only, and in the warmer parts of India the ascigerous stage is rare as compared with temperate areas like Kashmir.

In these cases, it is evident that the spore form which only appears at long intervals can be of little use to the fungus. It is able to live and reproduce itself for many generations by means of its other stages alone. Hence arises a gradual tendency to lose altogether the useless type of spore. A very large number of fungi are known which, from their relationships, we can guess to be conidial stages of Ascomycetes or sometimes of Basidiomycetes. To these the name of Deuteromycetes or imperfect fungi, is given, the term simply implying that they are suspected of being merely stages in the development of some higher fungus, but the higher form is not known, and is certainly in many cases altogether lost.

What adds to the difficulty of classifying these imperfect fungi is the fact that several different kinds of fungi (usually Ascomycetes) may all have the same kind of conidial forms. For instance, it is very common to find the conidium-bearing mycelium of *Fusarium udum* on stalks of pigeon pea which have been killed by the wilt disease. This mycelium bears two kinds of conidia on the same or different hyphæ. Occasionally, bright red receptacles, the sporophores of an Ascomycete, *Nectria Bolbophylli*, appear on the same stems, and by sowing the ascospores from these receptacles, we get a quite similar mycelium with two conidial forms almost identical with those of *Fusarium udum*. On the old roots and the base of the stem below ground a second kind of

Ascomycete, *Neocosmospora vasinfecta*, is sometimes found. This has a conidial mycelium very like the others and with quite similar conidia. Here, then, we have three entirely different fungi which resemble each other so closely in their conidial stages (which are often alone found) that we cannot readily tell, on examination of the conidial mycelium by itself which fungus we have before us.

### The dissemination of fungi.

A fungus may be disseminated by the mycelium or by spores. The former can usually only be carried with the substance on which it is growing, and sufficiently large pieces to contain portions of mycelium that will survive are not often carried far, except by the agency of man. Several parasites have, however, been widely distributed in the mycelial condition with the plants on which they grow, as, for instance, the well-known potato blight, which has reached many parts of the world in the tubers.

The great majority of fungi are ordinarily disseminated by spores. As these are very minute bodies (usually invisible to the naked eye) and are so light as to be easily taken up into the air as an impalpable dust, the wind is the chief agent in securing dissemination; there are, however, many subsidiary methods of spread.

The spores are often long-lived bodies, able to stand drying or extremes of heat or cold. The spores of bunt of wheat have been germinated after 8 years, and have been kept in a solid lump of ice for three months without injury, though the temperature fell to  $-20^{\circ}\text{F}$ . more than once. Other spores have been found to stand, when dried, the temperature of liquid hydrogen ( $-253^{\circ}\text{C}$ .) for several days. Susceptibility to heat varies greatly. Moist heat is more harmful than dry, and many spores cannot resist immersion in hot water at from  $120^{\circ}$  to  $150^{\circ}\text{F}$ ., for 10 minutes or so, while few can stand temperatures approaching the boiling point for even a short time. A dry heat above  $250^{\circ}\text{F}$ . is required to kill the spores of some species. So far from being injured by drying, most spores can usually be best preserved in a dry condition, though there are some exceptions, as in the sporangia of some of the Phycomycetes, which are killed by thorough drying for 24 hours. These properties of the spores greatly assist in the dissemination of fungi.

For dissemination by the wind, many fungi are peculiarly well-suited. They often produce spores in enormous quantities, up to 7 million million

having been calculated to be contained in the sporophore of *Lycoperdon bovista*, one of the puff-balls. When enclosed in receptacles, there are sometimes arrangements for expelling the spores forcibly into the air. These are often brought into play by changes in temperature or dryness, as in some *Ascomycoetes* where "puffing" (the expelling of a number of spores together in a cloud) can be induced by concentrating the sun's rays on the receptacle by means of a pocket lens. Many spores have tiny spines on the wall or are sticky on the surface; this helps to anchor them to leaves or other suitable places for germination. Others have thread-like tails or appendages, which doubtless assist both in keeping them in the air, and in fastening them to their support when they fall.

The distance to which spores can be carried in the air is not accurately known. There is no evidence that they can be borne for hundreds of miles (as is sometimes assumed) and much that they are often limited to a few miles. They are known to drop while in the air at rates varying from  $\frac{1}{2}$  to 5 mm. per second, and though the rate of fall is naturally influenced by vertical currents of air, in general they seem to be deposited much more quickly than might be expected at first sight. This is of great importance in dealing with the spread of parasitic fungi which, in a considerable number of cases under observation in recent years, have been found incapable of crossing the seas or of making large jumps within land areas, unless conveyed in some way by the agency of man. In many of these cases, the parasite has remained confined to a particular part of the world, until the opening up of new trade routes, the shortening of the time taken in transit, or the energies of Government departments, botanic gardens, and private individuals or trading firms in introducing exotic plants, have enabled it to travel to a new country on its host plant or in packings. Many parasitic fungi have reached Europe from North and South America and eastern Asia in this manner during the past 70 years, and new cases are reported every few years. The vine mildew, oidium, and black rot, the potato blight, the mildews of the gooseberry, oak, and *Enonymus japonicus*, the rusts of chrysanthemums, hollyhocks, and sunflowers, and many other parasites of more or less widely-grown plants, all appear to have been introduced to Europe in this fashion, while in no case recorded is there good presumptive evidence that they have travelled through the air from distant countries. Such long-distance or "discontinuous" dispersal as is going on at the present day, may apparently be always attributed to human agency.

Within a particular land area, as Europe or India, the dispersal of a newly-introduced species may be and often is "continuous," that is to say, the spread is gradual from plant to plant and long-distance jumps are not required. In many cases, there is evidence that extension may be checked by barriers of relatively small magnitude, if human agency does not assist the parasite to cross them. Thus the chestnut bark disease, which is at present exterminating the chestnut in the eastern United States, was unable for at least 10 years to cross a belt of mountainous country free from chestnuts, some 30 to 40 miles broad, to the west of the heavily-infected Hudson valley, after it was first introduced about 1904. In the same way, the blister blight of tea remained confined to the north-eastern corner of Assam for at least 40 years, though the disastrous outbreak in Darjeeling from 1908 showed it to be well adapted to other tea-growing areas. It has not yet crossed the mountains into Cachar, where the climate would seem to be suitable for it. In the palmyra palm disease, on the east coast of Madras, the attempt to check continuous spread during the past 10 years has been, on the whole, successful, though it would have been defeated could the parasite jump more than a few miles at a time. A palm more than 10 miles from one that is diseased seems to be safe from infection, and the usual jump seems to be less than 3 miles. The dispersal of the American gooseberry mildew in Europe seems to have been entirely due in the early days to distribution on diseased nursery stock from Denmark and Russia, to which it was imported from the United States. Norway, Sweden, Germany, Austria, and England were infected in this way, and the English outbreak was traced to imports on plants from the Continent, and not, as might have been expected, to spores blown through the air from Ireland, though Ireland had imported the disease from the United States several years earlier. In a general way, it may be said that, if human agency be excluded, dispersal within a new area is gradual and continuous; each new generation extends its area by perhaps some tens of miles; jumps of a hundred miles are rare, though they seem to have occurred in the case of the oak mildew in Europe; while jumps of several hundreds of miles are unknown.

For dissemination through short distances, the spores of fungi make use of many different methods of conveyance. Currents of air are by far the most important means in the great majority of cases. The splashing of rain and the flapping of wet leaves against one another is the chief means in a few cases, as in the bean anthracnose (p. 256), and is a secondary means in others, as in the potato blight (p. 277). Adhesion

by contact is well known to cause much of the bunt in wheat, when broken bunt balls get into the seed grain. Spread by insects takes place in certain well-established cases and is highly probable in others. The ergots of rye (*Claviceps purpurea*) and other grasses are carried in part by insects such as *Melanostoma mellina* and *Rhagonycha fulva*, which are attracted by the sugary secretion that accompanies the Sphacelia stage of the fungus. In *Sclerotinia urnula*, also, the sweet-smelling spores are carried by insects to the flowers of the cow-berry, which they infect. In many cases, insects which cause wounds allow parasitic fungi to enter, sometimes conveying them on their bodies. Larch canker may begin from the wounds of *Coleophora lariciella* and *Chermes laricis*; *Ceratostomella pilifera* (a fungus which causes blueing of the wood of *Pinus ponderosa*) from wounds of *Dendroctonus ponderosae*; while the Nectrias, which cause cankers of fruit and forest trees, are said sometimes to be conveyed on the bodies of insects which cause wounds through which the fungi can enter. Occasionally, other animals have been accused of disseminating parasitic fungi; mice or rats are said to carry the spores of *Fomes annosus*, the cause of a well-known pine disease, and snails or slugs those of some parasites of fruit trees and vegetables (vine mildew, Nectria, *Bacterium campestre*). Non-parasitic fungi are also sometimes disseminated by animals, as the Phalloids whose spores are largely carried by flies attracted by their strong smell, and the truffles which are eaten by pigs and other animals and whose spores survive digestion.

The large group of soil-inhabiting fungi, which do not often produce aerial sporophores, are liable to be carried by irrigation water, or by surface wash after heavy rain; *Fusarium* is undeniably carried from field to field by the latter, as we know from the spread of the *Fusarium* wilts. Infected soil may also carry the spores of such fungi and may reach other fields in various ways, as on the boots of farm workers and pedestrians, or on agricultural implements. All these and other readily conceivable minor methods of spread are effective only for relatively short distances. The flight of insects, the flow of irrigation water, or the range of labourers and pedestrians, is usually through a limited area; dissemination through the air may be wider but is still restricted; and though we could imagine diseases crossing land frontiers or even narrow seas in this manner, we have to fall back on human transport to explain the introduction of fungi from distant parts of the world.

How effective human agency may be in introducing fungi may, perhaps, best be grasped by examining the fungus flora of the great botanic

gardens, such as those of Kew or Berlin. In these gardens, a large number of foreign species appear from time to time and are unquestionably introduced with plants or packings from abroad. Kew, for instance, has a much larger recorded fungus flora than any other area of similar size in the world. It is the somewhat tardy recognition of this fact which forms the base of all the modern restrictive legislation against the free circulation of plants, which most civilised countries have framed to protect their agriculture against the introduction of new fungus diseases.

### CLASSIFICATION.

The fungi as a whole are divided into three classes: Phycomycetæ, Ascomycetæ, and Basidiomycetæ. To these must be added, as an appendix, the Deuteromycetæ, or imperfect fungi, which, as already stated, are conidial fungi, in many cases forming a stage in the development of Ascomycetes or Basidiomycetes, but in the majority existing only as conidial forms, having lost their higher stages.

### OUTLINE OF CLASSIFICATION.

#### LOWER FUNGI.

*Hyphæ not interwoven to form compact tissues, not regularly septate.*

**Class I.—Phycomycetæ.**—Sexual reproduction distinct, the process being usually completed within the sexual cells themselves.  
Asexual reproduction by sporangiospores or conidia

Sub-class I.—Oomycetæ.—Sexual reproduction by oospores

Order I.—Chytridiaceæ.

„ II.—Monoblepharidaceæ

„ III.—Leptomitaceæ.

„ IV.—Ancylistaceæ.

„ V.—Saprolegniaceæ.

„ VI.—Peronosporaceæ.

Sub-class II.—Zygomycetæ.—Sexual reproduction by zygospores.

Order I.—Mucoraceæ.

„ II.—Entomophthoraceæ.

#### HIGHER FUNGI.

*Hyphæ usually interwoven to form tissues at some stage in growth, especially in the sporophore, septate.*

**Class II.—Ascomycetæ.**—Sexual reproduction more obscure, often in two phases, the sexual nuclei uniting in a cell derived from that in which they first came together, or sometimes reduced to the union of sister nuclei; resulting in the production of ascospores. Asexual reproduction by conidia.

Order I.—Gymnoascaceæ.

„ II.—Pyrenomycetaceæ.

„ III.—Discomycetaceæ.

„ IV.—Laboulbeniaceæ.



**Class III.—Basidiomycetes.**—Sexual reproduction waning and reduced to the union of sister cells or even of sister nuclei ; resulting in the production of basidiospores. Asexual reproduction by conidia.

**Sub-class I.—Hemibasidia.** Basidia septate or not, often irregular in growth, basidiospores irregular in number, usually forming secondary spores on germination.

Order.—Ustilaginaceae.

**Sub-class II.—Protobasidia.** Basidia of more strictly limited growth, septate into usually four cells each bearing only one basidiospore.

Order I.—Uredinaceae.

„ II.—Tremellinaceae.

**Sub-class III.—Eubasidia.** Basidia of strictly limited growth, aseptate, bearing a definite number of basidiospores, usually four.

Order I.—Hymenomycetaceae.

„ II.—Gasteromycetaceae.

**Appendix.—Deuteromycetes** (imperfect fungi). Asexual reproduction by conidia. Some are known to be conidial stages of higher fungi.

Order I.—Sphaeropsidaceae.

„ II.—Melanconiaceae.

„ III.—Hyphomycetaceae.

The first or lowest class of fungi is the **Phycomycetes** or alga-like fungi. These include the aquatic fungi and the simplest land fungi. Their hyphae never unite into strands or tissues, and the mycelium is either filamentous or composed of isolated, rounded cells. Regular septation is never found, so that the living parts of the hyphae usually form a continuous cell. The characteristic asexual fructification is the sporangium. Conidia are also found in some orders. Sexual reproduction is of a distinct and simple type, and the nuclei usually fuse in the same cell in which they come together. They are divided into two sub-classes, the Oomycetes and the Zygomycetes.

The **Oomycetes** have their sexual reproduction by female cells known as oogonia, which are fertilised by male antheridia to form oospores. They are divided into six orders :—

**Chytridiaceae.**—Usually one-celled fungi of very simple structure ; the whole body in many cases consists of a rounded cell which becomes directly transformed into a sporangium. The sexual reproduction is variable in type and often unknown. No conidia are known. Mostly parasites in water-plants, less often in land-plants.

**Monoblepharidaceae.**—Aquatic fungi with sexual reproduction in which the oogonium is fertilised by small, swimming, zoospore-like bodies, termed antherozoids, formed within antheridia. Asexual reproduction by zoospores from sporangia. Conidia not known.

**Leptomitaceae.**—Aquatic fungi with the hyphae constricted at intervals and nearly closed by partitions, but not truly septate. Sexual reproduction by the union of oogonia and antheridia to form oospores : only known in some cases. Asexual reproduction by zoospores from sporangia. Conidia not known.

**Ancylistaceae.**—Aquatic fungi, parasitic in algae and small water-animals. Resemble the *Chytridiaceae* in their reduced mycelium and the *Leptomitaceae* in its constriction into segments. The whole of a segment becomes an antheridium or oogonium or an asexual sporangium with zoospores.

*Saprolegniaceae*.—Aquatic fungi with often large hyphae, usually saprophytic. Sexual reproduction by antheridia and oogonia, each oogonium usually bearing a number of oospores. In most cases no actual fertilisation occurs. Asexual reproduction by zoospores, usually from elongated sporangia. Conidium-like bodies are sometimes formed from the sporangia by suppression of the zoospores.

*Peronosporaceae*.—Chiefly land-fungi parasitic on plants, a few also aquatic and saprophytic. Sexual reproduction by oogonia and antheridia, fertilisation resulting in a single oospore. Asexual reproduction chiefly by zoospores from rounded sporangia. Conidia often formed from the sporangia by suppression of the zoospores.

The *Zygomycotaceae* have their sexual reproduction by the union of two more or less similar cells to form a zygospore. The asexual reproduction is never by zoospores. They are divided into two orders :—

*Mucoraceae*.—Land fungi, chiefly saprophytic. Zygospores formed by the union of two usually equal cells. Asexual reproduction typically by rounded air-disseminated spores formed in a sporangium, rarely also by conidia.

*Entomophthoraceae*.—Parasitic on insects, rarely on plants or saprophytic. Sexual reproduction by zygospores formed by the union of two often unequal cells. Asexual reproduction by conidia only.

The *Ascomycetes* include a great number of forms, all agreeing in having spores developed within a mother-cell, the ascus. In most, the asci are produced on, or in special sporophores; these are known as perithecia when round or flask-shaped and enclosing the asci in the hollow cavity, and as ascophores or apothecia when the asci are exposed. Conidia of the most diverse sorts are also usually borne. The mycelium is, on the whole, less aggregated into masses than in the *Basidiomycetes*; in the majority of cases, however, the asci are developed on a hymenium lining a sporophore formed by the union of many hyphae, and in some there is a considerable development of stromatic tissue in which a number of perithecia may be borne (compound stromata). Besides these sporophores, aggregations of hyphae to form sclerotia are found at times. The asci are usually of regular size, with eight spores. Sometimes there are less than eight spores, sometimes more, up to sixteen, thirty-two, sixty-four, or more. There are four orders :—

*Gymnoascaceae*.—Asci formed directly on the mycelium or on special parts of it, not united in a hymenium enclosed in or borne on a complex sporophore. In the *Eozascaceae*, which are parasites, the asci are formed under the surface of the leaf, becoming exposed when ripe. In *Endomyces* they occur singly as lateral outgrowths of the hyphae. In *Gymnoascus* there is a rudimentary perithecium, formed of a loose web of branched hyphae, within which the asci are entangled. The yeasts, or *Saccharomycetaceae*, differ from all the other higher fungi in having a much reduced gemmate mycelium, consisting of a single cell which buds off other similar cells or may become converted as a whole into an ascus; in some cases, at least, the formation of the ascus is preceded by a sexual act in which two cells and their contained nuclei fuse.

*Pyrenomycetaceae*.—Asci formed usually on a hymenium lining the interior of a hollow sporophore (perithecium), which is usually flask-shaped and with a narrow mouth through which the spores escape at maturity. The perithecia are variously arranged, sometimes occurring independently on the mycelium, in others joined together into groups in compound stromata, which may be crust-like or tubercular or erect and even stalked. In the edible truffles (*Tuberaceae*), the sporophore is an underground tuberous body with one or more internal chambers, or a series of labyrinthiform passages, lined with the hymenium. Conidial forms are extremely common and varied.

*Discomycetaceae*.—Aci formed on a hymenium exposed on the surface of the ascophore. The latter is usually cup-shaped with incurved margins, which unfold as it matures, exposing the hymenium. Compound stromata are rare but in some cases, as in the edible morels, the sporophore has a distinct stalk and the hymenium lines the outer convoluted surface of the fertile head, which may be of considerable size.

*Laboulbeniaceae*.—A peculiar group of fungi, found on the softer outer parts of insects, where they form tiny, stalked outgrowths, usually less than one-twentieth of an inch in length. The whole body consists of a very few cells arranged in a manner not found in other fungi, a group of cells of very different sizes, shapes, and functions being formed at the end of a stalk cell; some of these are male organs, bearing antheridia which give off small antherozoids; others are female cells—oogonia—with a special receptive organ called a trichogyne at the top of each. The antherozoids unite with the trichogynes and perhaps pass down to fertilise the oogonium below. The latter then divides into several cells, some of which give rise to the asci. The ascus produces four or eight ascospores.

The **Basidiomycetes** include a very large number of the higher fungi, representing the most diverse types. All, however, have the character that at some period in their life-history, following the completion of a sexual act, spores are formed on sterigmata produced from special, fertile cells, the basidia. They are divided into three sub-classes, Hemibasidiales, Protobasidiales, and Eubasidiales.

The **HEMIBASIDIALES** include only one order, the *Ustilaginaceae* or smuts, parasites which attack grasses and other plants and produce spores, as a rule, only in certain organs of their hosts, generally the flowers. These spores usually form a dense black dust, from which the name "smuts" is taken. On germination, they give out a promycelium or short germ-tube, which is a transitional form between the ordinary type of germ-tube and the basidium of the other two sub-classes. It is either transversely septate or aseptate, and each cell bears a variable number of sporidia, on sterigmata or sessile. On germination, these may bud off secondary spores. The vegetative mycelium is filamentous, and there is no formation of compact tissues except sometimes in the spore-beds.

The **PROTOBASIDIALES** differ from the Hemibasidiales chiefly in having a more regular basidium and a definite number of sporidia, each cell of the usually 4-celled basidium producing a single sporidium on the end of its sterigma. They are divided into two orders:—

*Uredineae* or rusts.—True parasites of green plants. Spores produced in cup or flask-shaped or flat spore-beds and of five different kinds: spermatia, aecidiospores, uredospores, teleutospores, and sporidia. One or more of these may be absent. On germination, the teleutospore produces a promycelium like that of the smute, but more strictly limited in growth. This represents the basidium and from each of its (usually four) cells a single sporidium is produced on a sterigma. The mycelium is filamentous, except where it condenses to form the spore-beds.

*Tremellinaceae*.—Saprophytes with the hyphae united into masses, usually gelatinous when moist. The basidia are produced generally on fertile hyphae joined into a hymenium, which may be either superficial or enclosed by a protective covering of sterile hyphae. They are septate, usually into four cells, each of which produces a single basidiospore. On germination, conidia may be produced almost immediately ("secondary spores") or they may be formed on the mycelium at a later stage.

The **Euascomycetes** include the mushrooms, puff-balls, and most of the larger fungi. The hyphae frequently unite to form large, conspicuous masses, particularly in the sporophores, but also in sclerotia and rhizomorphs. The basidia are generally joined together into a hymenium and are acutate, with a definite number of basidiospores, usually four, borne on sterigmata. Conidia are found in some cases. They include two orders:—

*Hymenomycetaceae*.—Saprophytes or parasites. Basidia forming a hymenium, which is exposed from the first or at least before the spores are ripe.

*Gasteromycetaceae*.—Chiefly saprophytes. Basidia forming a hymenium, which is enclosed in the sporophore by a special covering of sterile hyphae until after the spores are mature.

The **Deuteromycetes** comprise a large number of conidium-bearing fungi, some of which have been proved to be merely stages in the life-history of Ascomycetes, more rarely Basidiomycetes, or very occasionally Phycomycetes. The vast majority, however, appear to exist habitually as independent individuals, having probably lost any other method of reproduction than that by conidia. They are entirely devoid of sexual reproduction or sporangium-formation, and are usually of minute size. Many are parasitic. They are divided into three orders:—

*Sphaeropsidaceae*.—Conidia borne on a hymenium, enclosed in a round, flask-shaped, or flattened sporophore (pycnidium or pseudo-perithecium). The conidia are set free through a narrow mouth or a slit.

*Melanconiaceae*.—Conidia borne on crowded conidiophores arising from an immersed stromatic base, in a cavity, of which the outer wall is formed by the tissues of the host (acervulus).

*Hyphomycetaceae*.—Pycnidia and acervuli absent, conidiophores superficial, sometimes produced from simple free branches of the mycelium, at others, arising from an erect bundle of hyphae united together into a strand, or from a wart-like superficial cushion of matted hyphae or of stromatic pseudoparenchyma.

---

## CHAPTER II.

### THE FOOD OF FUNGI.

THE spore, on germination, gives rise to a germ-tube, which at first is nourished by reserve food stored up in advance. In many cases, a large proportion of this food is fatty in nature, and is visible in the form of oil or fat droplets in the ripe spore. Very soon the young hyphæ begin to feed for themselves. As in the case of other plants, the food must be dissolved before it can be taken in. This is necessary because solid particles cannot pass through the membrane which forms the cell wall of all hyphæ. Liquids can, however, pass through, and the food dissolved in them reaches the interior of the hyphæ by a process of osmosis.

The great difference between fungi and the plants which possess the green matter called chlorophyll, is that the former cannot build up their essential carbonaceous food out of the carbon-dioxide of the air. In this respect the fungi are like animals, requiring their food ready prepared by having been built up to form the bodies of other plants or animals.

Just as many animals require a certain amount of mineral food, such as common salt, for their perfect health, so also mineral salts are necessary to most fungi. Amongst the chief minerals important in this respect are potassium, magnesium, and perhaps iron. Such small quantities of these are required that sufficient is generally obtainable in nature from the soil, from decomposing vegetable or animal matter, or from the sap of living plants. To be available as food they are best dissolved in solutions of weak strength.

The other important constituents of the food of fungi are, as in the higher plants, nitrogen, phosphorus, and sulphur.

According to the manner in which fungi obtain their organic food, they are divided into two great classes :—Saprophytes and Parasites.

**SAPROPHYTES** obtain their organic food from the dead tissues of animals or plants, or of substances derived from them. Hence they are found in large numbers in the soil, pervading every bit of rotting leaf

or twig or particle of manure. They grow as moulds on fruit and jam and on boots and the backs of books in the rains. Every bit of old timber in the forests is liable to be permeated all through with their hyphæ. In company with the bacteria, they play the part of scavengers, breaking up such resistant organic bodies as the walls and contents of plant cells, liberating carbon-dioxide which becomes available in the air as food for green plants, hydrogen which soon forms water, ammonia, and so on.

PARASITES obtain their food from the living tissues of animals and plants. While, therefore, many saprophytic fungi play a useful part in nature as scavengers, the parasites are almost entirely hurtful. The few cases of beneficial parasites, from the point of view of man, are those which attack noxious insects and occasionally destroy large numbers of them. The great majority of parasitic fungi feed on living plants, those useful to man as much as, or more than, the weeds or useless kinds. A living plant on which a fungus feeds is often termed a "host plant" for that fungus. There is scarcely a plant of our fields, gardens, and forests, which does not serve as a host plant for one or more species of fungi.

Several different classes of parasites may be distinguished. *True parasites*, for instance, pass through the whole of their life-history on living plants and cannot be grown on dead or artificial food material. Such are the rusts and some Ascomycetes and other fungi. *Hemi-parasites* are those that feed usually on living tissues but can, at need, pass through a part of their lives as saprophytes. Often their full development is only reached on the living plants and we cannot by artificial cultivation get them to complete the whole of their life cycle. The smuts are examples of this, as they can often live by budding out little gonidia for a considerable time in soil, but only grow to maturity and produce their perfect spores on a living plant. *Hemi-saprophytes*, on the other hand, are those which usually grow on dead or decaying matter and are capable of passing through the whole of their development as saprophytes, but have at the same time the faculty of attacking living tissues under certain conditions.

Within these main divisions, parasitic fungi may show every degree of adaptation to the parasitic existence. At the lowest end of the scale are certain hemi-saprophytes, which may be called "weak parasites," as they become parasitic only occasionally, when the host plant has been weakened in its vitality by some harmful agency. Plants not yet acclimatised in a new locality, or suffering from insect attack, or grown in insanitary conditions such as overcrowding, too dense shade, unsuitable

soil and the like, are particularly liable to become available as food to these fungi that are normally saprophytic. Cases of the sort have been recorded from time to time, though few have been adequately investigated; for instance, exotic cottons which are being unsuccessfully acclimatised in India are often invaded by a common species of *Alternaria*, which is able to overcome the lowered vitality of the cells of the leaf and cause their death, though acclimatised or indigenous varieties are not attacked by it and it usually seems to be a pure saprophyte.

Instead of this weakened vitality of the host plants aiding a normally saprophytic fungus to live on them, certain saprophytes have been found at times to become "educated" into the parasitic life; that is to say, it has been found possible to develop a parasitic strain, or race, of a normally saprophytic species, capable of living even on healthy individuals of the host plant. Whether such a strain might become permanently fixed in the parasitic life is not known, but it seems probable that parasitism as a whole must have originated in some such manner. The best authenticated instances of this kind are in the bacteria, where it is well known that virulence may vary greatly in different strains of the one species. Thus the common *Bacillus coli* has been found unable to attack strong, well-grown, mature potato tubers, when it is first cultivated as a saprophyte on dead substances, but if the tubers are weakened in their resistance by growth in an excess of lime, or by soaking in weak alkali, they can be readily infected, and the strain thus produced is actively parasitic when directly inoculated into unweakened tubers. Amongst fungi, the saprophytic *Cladosporium epiphyllum* has been reported to have developed into a parasite on a variety of *Clerodendron fallax*, in which the leaves were studded with glands, on the sugary secretion from which the fungus grew at first as a saprophyte. Having become accustomed to the secretion of the gland cells, it proceeded to attack the cells and ultimately became capable of infecting any part of the leaf. Other similar cases have been recorded, as for instance where the saprophytic *Trichothecium candidum* was artificially trained into a parasitic life on begonia leaves, by injecting sugar into the leaves. Still more frequently it has been observed that the opposite may occur, and a parasite may lose the power of living on its host plant by being artificially grown for several generations as a saprophyte.

In a somewhat more developed stage of parasitism are those cases where a fungus is a weak parasite on some hosts, while on others it can

attack normal healthy plants. *Thyridaria tarda*, described below as a parasite of sugarcane, tea, and other plants, seems to be a case of this kind.

In many cases, particular tissues of a plant are normally of low vitality and are readily invaded by weak parasites. Such are the gorged cells of many pulpy fruits, oranges, mangoes, apples, and the like, and also the older wood of trees.

Next, there is the large section known as "wound parasites." At first these live only on the dead and rotting tissues caused by a wound or injury to the plant. Later on they begin to spread to the undamaged cells further in, and they may extend through a large area of the living tissues, eventually, perhaps, killing the whole plant. Wound parasites are very common on trees, as the large masses of dead bark and wood which result from the injuries to which trees are liable (as when a branch is torn off by the wind) enable the fungus to get a firm footing before it proceeds to attack the living tissues. A large wound may also expose the older wood which, as already mentioned, is particularly liable to attack in some cases.

Other hemi-saprophytes are more vigorous, being able to attack perfectly sound tissues of healthy plants should they come in contact with a suitable host, but in the absence of the latter can live and develop freely on dead material. Such a case is the "damping off" fungus, *Pythium de Baryanum*, which is present as a saprophyte in almost every garden in some parts of Europe, and at once attacks and destroys seedlings of cress and certain other plants, should they be grown in the soil which contains the fungus. The species of *Fusarium* that produce wilt diseases can also live in the soil as saprophytes for considerable periods, but will attack suitable host plants in such soil when the latter are otherwise healthy and growing under ideal conditions for their development. Neither neglect of cultivation nor poverty of soil predispose to these diseases; wounds are not necessary for the entry of the parasite; and, if anything, wilt is worse on highly cultivated and well fertilised fields than elsewhere.

Even amongst the hemi-parasites and true parasites there are different degrees of adaptability to the parasitic life. Some are able to pass through all the tissues of the host plant, others are confined to certain tissues or certain localities on the plant. In several cases, the parasitic mycelium is only capable of growth in young or embryonic tissues. In some of the cereal smuts, for instance, the mycelium cannot



continue its development unless it early reaches the multiplying cells of the growing point; otherwise it ceases growth and gradually disintegrates. In *Peronospora parasitica* too, the whole plant is sometimes invaded, but this usually occurs only while it is still composed of young tissues as in the seedling stage; at other times, chiefly in older plants, the attack is local and confined to parts where the cells are still dividing, as in the young inflorescence, or the stem galls caused by *Cystopus candidus*. In maize smut, local infection is the rule, and none but young tissues are involved. Similarly, several of the parasitic Phytophthoras can only attack young individuals of certain hosts, and the same is noticeably true for the peach and almond mildew (*Sphaerotheca pannosa*) in Kashmir, where the full-grown trees are almost immune though nursery stock suffers severely.

It has been established in several cases that external conditions may have a considerable influence in promoting or checking the growth of the parasite in the plant. Temperature, humidity, and conditions affecting the nutrition of the host, are amongst the most important factors in these cases. Thus, in some of the cereal rusts, the period of vegetative existence of the uredo stage may be much prolonged at certain times of the year. In India, the uredo stage of black rust of wheat may be prolonged, in the hot weather, from the usual 10 or 15 days to 35 days, and, in other countries, infections in the beginning of winter have not resulted in spore production until the following spring. The same has been found to occur in the teleuto stage of the hollyhook rust. Again, it has been proved possible to check the growth of the uredo mycelium in some cereal rusts, after infection has been successfully accomplished, by interfering with the nutrition of the leaf, such as by starving it of carbon-dioxide, or by heating or cooling the roots and so preventing them from carrying out their functions properly. If interference with the plant is carried too far, the mycelium will die, but in some cases it is only arrested and resumes growth later on. It is probably for the same reason that it has not been found possible to get successful inoculations with some parasites, unless the host is in a healthy and vigorous condition. *Cystopus candidus* has been observed not to grow on host plants infested with aphids or thrips, or if the young leaves are unhealthy and yellowish, and the same is true of the cucurbit mildew, *Erysiphe Cichoracearum*. Of the downy mildews generally, it has been said that the more healthy a plant is, the more *Peronospora* appears to prosper on it. Nevertheless, far more cases are known where the opposite is true; all the weak parasites are favoured by lowered vitality, deficient nutrition, or any unhealthy state

of their hosts, and even the more highly developed parasites are often quick to take advantage of any weakening in the powers of resistance of the host. One of the best cases of this kind recorded is in maize smut, where, by exposing the plant to the vapour of ether, or heating it to 70°C, total and rapid invasion has been induced, though ordinarily the fungus is strictly local in its attack. Very many instances will be found in the part dealing with special diseases below, in which weakened vitality results in increased severity of the attack, and it is not necessary to delay longer to consider specific cases.

The parasitic fungi may further be divided into two types, "ecto-parasites" and "endoparasites," according to their situation on their host plants.

ECTOPARASITES include those forms which grow on the surface of the leaves, stems, or other parts of the affected plant, and obtain their food through the outer cell walls without penetrating deeply into the tissues. They feed usually by means of special outgrowths from the hyphæ, known as suckers or "haustoria." These arise from hyphæ in contact with the epidermis, and penetrate wholly or partly through the toughened outer walls of the epidermal cells. All the body of the fungus lies outside the plant, excepting these haustoria, and there are even several cases where haustoria have not been detected and it seems as if the food is absorbed directly from the outer cells across the unbroken cell wall. Hence the whole or practically the whole life of these parasites is passed externally, and this is of significance when the question of destroying them comes to be considered.

ENDOPARASITES, on the other hand, penetrate into the plant and develop their vegetative mycelium within the tissues. Sometimes the internal mycelium is confined to a small part of the plant, as in the case of the numerous leaf-spotting fungi, where each little patch of invaded leaf tissue shows as a discoloured spot owing to death of the cells. When a single spore of one of these leaf-spotting fungi germinates on a suitable leaf, the germ-tube penetrates into the leaf, grows, branches, and the resulting hyphæ spread all around in the tissues. Their food is obtained from the cell sap of the living leaf cells, either by the hyphæ or suckers from them growing directly into the cells in search of it, or by absorption through the walls in the case of such fungi as live between, not inside, the attacked cells and which have no haustoria (e.g., *Cercospora longipes*). When a certain amount of food has been obtained, and a certain number of leaf cells killed, the fungus ceases its vegetative growth and reaches

the reproductive stage. In other endoparasites, the growth may extend to a considerable part of the plant (e.g., *Phytophthora infestans* and many parasites of stems and roots), and reproduction may occur while vegetative growth is still going on. The formation of spores within the tissues would be of little use in disseminating the fungus, so the endoparasites practically all send out special sporophores to the surface of the plant, and form their spores in the outside air. In some cases (e.g., Peronosporaceæ) a second kind of spore, usually a sexually-formed resting spore, is developed within the tissues, and this is only set free and germinates when they have rotted. The use of this second spore form is generally to carry the fungus over some unfavourable season, so that germination will only occur when conditions are again suitable for the general life of the fungus.

### Mechanism of feeding by ectoparasites.

THE simplest arrangement by which parasitic fungi reach their food is found in such ectoparasites as cover the surface of leaves and other parts of plants with a web-like mycelium, which may be lifted off, leaving the surface bare and the walls of the outer cells intact. The fungus which causes the thread blight of tea is an instance of this. The parasite forms white strands which lie on the stems and twigs of the tea bush, and spread out fan-wise in a thin, glistening, membranous layer, over the under surface of the leaves. This membrane may be peeled off quite unbroken from moist leaves, and careful examination shows that the outer walls of the leaf cells under the membrane remain intact. Leaves that are thus covered soon begin to droop and wither as a result of the death of the leaf cells. At the same time the fungus is evidently plentifully supplied with food and is able to continue growth and to send fungus strands across to neighbouring leaves. Every patch of leaf which the membrane reaches undergoes these changes; and as the fungus does not interfere with light and is not dense enough to check the passage of air to any serious degree, the effects are doubtless due to an absorption of food from the living leaf by the fungus, possibly preceded by the secretion of some substance (toxin) capable of killing the leaf cells. There are no special suckers, or haustoria, penetrating into the cells, and so the food must be taken by absorption across the unbroken walls of the leaf cells and fungus hyphæ by endosmosis. It is probable that the fungus (*Corticium (Pellicularia) Koleroga*) which causes the koleroga disease of coffee in Mysore, acts in just the same manner, as here also no haustoria have been found.

In the ectoparasite of certain European pine trees, *Acanthostigma parasiticum*, a white mycelium forms cushion-like layers on the under surface of the pine needles, and obtains its food by tiny, rod-like haustoria, which grow from the lower hyphæ and indent the outer walls of the leaf cells, passing only into the outermost layer of these, the cuticle (Fig. 15, 1). In this case also, food is taken in by the parasite by endosmosis across a part of the wall of the leaf cell, into the special portions of the hyphæ (the haustoria) which are formed for the purpose of obtaining it.

In the fungi known as Erysiphaceæ, or mildews, most of which are ectoparasites, the mycelium forms a white mould on the leaves, from the inner hyphæ of which haustoria are formed and grow through the cell

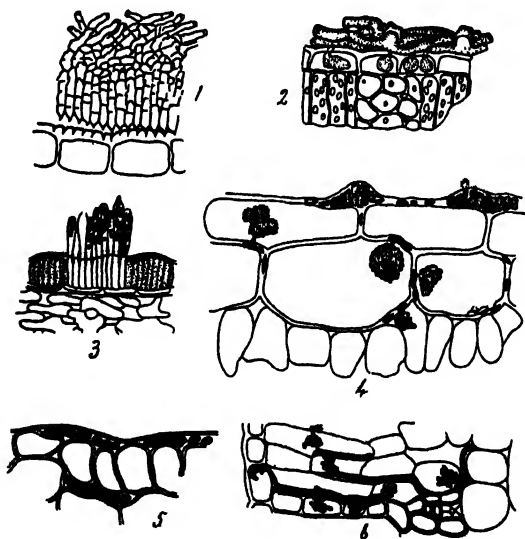


FIG. 15. 1, ectoparasitic mycelial cushion of *Acanthostigma parasiticum*; 2, ectoparasitic mycelium of oak mildew; 3, mycelium and aeci of *Taphrina rhomboidalis*; 4, mycelium and haustoria of *Taphrina masulana*; 5, mycelium of *Eucosmoua deformans*; 6, mycelium and haustoria of *Cercospora personata*. (1 after Ducomet, 2 after Ferraris, 5 after Pierce.)

wall into the interior of the cell cavity (Fig. 15, 2). Inside the attacked cell, the haustorium swells up into a bladder-like, simple or branched sac, with thin walls, into which the sap of the living cells is absorbed and

passes back to the mycelium outside. It is evident that these mildews have discovered a more satisfactory manner of getting at their food than the fungi mentioned above, as, by forcing their suckers right into the living cells, they break down the protection which is afforded by the cell wall and come into direct contact with the living cell contents.

### Mechanism of feeding by endoparasites.

A few endoparasites live in the membranes of the outer cell walls of their host plants. Such are some of the *Exoascaceæ* in which the mycelium lives under the outer cuticle of the cell wall (Fig. 15, 3). This position it reaches by the germ-tube of the spore growing from the surface of the leaf in under the cuticle, and forcing for itself a passage between the cuticle and the true cell wall. There are no haustoria and the fungus feeds by osmosis through the part of the cell wall intervening between it and the cell cavity. When spore formation occurs, the cuticle is ruptured and the fungus, which up to then was completely immersed, becomes exposed to the air.

In some other members of this group, the mycelium penetrates between the leaf cells but does not enter into their interior. Haustoria are found in one species, *Taphrina maculans* (Fig. 15, 4), but are absent in the others. Sometimes the hyphæ are confined to the outer layers of cells, sometimes they spread through the whole leaf, passing either in the intercellular spaces which are found at the angles of many cells, or forcing their way between the walls of adjacent cells. The latter condition is found in *Exoascus deformans*, the cause of the peach leaf-curl so common in the hills (Fig. 15, 5). In these cases, the hyphæ obtain their nourishment by endosmosis from the cells outside of which they lie.

A large number of fungi resemble the last in sending their vegetative hyphæ between the cells of the attacked plant in all directions, but obtain their food by haustoria which are pushed directly from these hyphæ into the interior of the cells (Fig. 15, 6). These haustoria are often of characteristic shape, some being simple stud-like sacs, others clumped masses somewhat resembling a bunch of grapes, and others finger-like or with branches that may fill a great part of the cell (Fig. 16).

Still another—and the largest—group of endoparasites, send their hyphæ not only between, but also directly into, the attacked cells. As a rule these parasites are more rapidly destructive than the others, for they generally kill all the penetrated cells. The haustorium-forming fungi, on the other hand, often seem to exist for a long period on some part of

the cell sap not essential to the life of the cell, or absorb their food so slowly that the cells are able to provide sufficient to feed the fungus

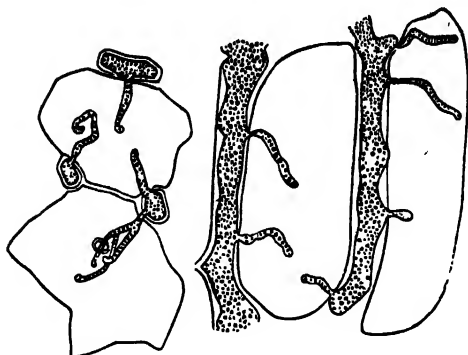


FIG. 16. Haustoria of *Sclerospora graminicola* on left and *Phytophthora Colocasiae* on right.

without suffering any great damage themselves. In most of these cases, however, the period when the parasite begins to form sporophores and spores on the surface is one of such activity on its part, and requires so much food for its attainment, that the infected cells are killed, and the part containing the fungus withers.

In very many species, active parasitism is confined to the earlier part of the life-history; the later stages, particularly the development of the "perfect" spore form of the fungus, occur only after the tissues of the host plant have been killed. Thus, in many Peronosporaceæ and Ascomycetes, the oospores, or the perithecia, only appear on dead parts, and the food requisite for their production is obtained either from the dead organic matter of the cells or, more probably, from reserves stored up in the mycelium during its period of parasitic activity.

#### Preparation of the food for absorption.

In the above pages, some of the measures taken by fungi to reach their food have been described. The saprophytes have no difficulty in this, for their young hyphæ can absorb food from the substance on which they are growing. The parasites may similarly absorb food through the surface walls of the living plant, but generally have to make some

arrangement for entering into its tissues, either directly, by means of ordinary hyphae, or through special organs, the haustoria. Having arrived in contact with the food, their work is not, however, finished. Very often the cell substances are present in an unsuitable form and must be altered to enable them to serve as nutrition for the fungus. Starch, for instance, which is present in so many vegetable cells, has to be changed to sugar (e.g., *Polyporus sulphureus*), and cane sugar has sometimes to be inverted to glucose (*Ustilago Zeae*, *Colletotrichum falcatum*); living protoplasm has to be killed and altered into other nitrogenous compounds, such as peptone; glucosides have to be split up to liberate carbohydrates, such as glucose, which may serve as food (several fungi parasitic on trees); fats are also probably split into glycerine and fatty acids. In those fungi which make use of the cell membranes, cellulose and pectic bodies have to be dissolved. These processes are carried out by means of ferments, in which fungi, both parasitic and saprophytic, are exceedingly rich. Ferments (enzymes) capable of causing the alterations mentioned above, and several others, have been found to be commonly produced by fungi and must play an important part not only in transforming the raw food material into products suitable for absorption but also in making use of the food taken in. In the symbiotic forms, and many of the more highly organised parasites, such as some of the smuts, the fermentative activity is so controlled that the cells of the host may suffer little, or not at all. In other cases, however, the first action of the parasite is to kill the living cell contents, and this may occur even before the hyphae have actually penetrated through the cell wall, though the latter seems usually to be partially dissolved by cellulose or pectin-dissolving ferments before the toxic action of the fungus on the living contents becomes evident: whether the toxic substances are the same as those which dissolve the cell wall (p. 95), or are different, is not yet fully known, but in some cases (*Botrytis cinerea*) there is evidence that they are identical or at least chemically allied. Intermediate between these two extremes are the cases in which the parasite causes an irritation or stimulation of the tissues and provokes more or less deep-seated alterations in the nature and disposition of the cells, leading to the galls, deformities, and other conditions more fully discussed below (p. 84).

The substances of the host plant utilised for food vary greatly with different parasites. Not alone are the latter often confined to certain tissues, but some attack chiefly the cell membranes, others the cell contents. The Peronosporaceae and rusts, for instance, often consume little or nothing of the membranes, using chiefly the cell contents;

various parasites of woody tissues (where the cell contents are often scanty) get most of their food from the walls, leading to the honeycomb-structure, or the large cavities, found in the older wood of diseased trees ; while in a few cases (e.g., *Sclerotium rhizodes*) cell contents and cell walls are equally destroyed. Within the cells, some consume the starch (*Phytophthora "omnivora," Claviceps purpurea*), others seem to leave it alone (*Guignardia Bidwellii, Pythium de Baryanum*) ; sugar is diminished in sugarcane infected by *Colletotrichum falcatum* ; many vigorous parasites rapidly destroy the nitrogenous cell matter ; while in such forms as the rusts, which have so far defied artificial cultivation, there is evidently something essential which has not been successfully isolated as yet from the living cells. Even such poor food materials as tannin and aromatic bodies are utilised by some fungi (e.g., *Trametes pini, Endothia parasitica*, and probably the fungus which causes the formation of "brown oak"). In dealing with the effects of parasitic fungi on the cells of their host plants (p. 93), this subject will be further considered.

Amongst the ferments most commonly found in fungi, are diastases, which transform starch into sugar ; invertase, which inverts cane sugar ; proteases, which render insoluble proteids soluble ; cytase, pectinase, cutinase, and hadromase, which attack cellulose and modified cell walls ; lipase, which splits fats ; emulsin and allied ferments, which decompose glucosides. The single fungus *Polyporus squamosus* has been found to form diastase, protease, lipase, emulsin, tyrosinase, laccase, rennetase, and "coagulase," while evidence was obtained that it also produces cytase and possibly hadromase. What the toxic substances are which kill the living protoplasm, or cause the formation of galls or other abnormalities, is not known.

### Effects of different kinds of food.

ALL saprophytes and many parasites can be grown on artificially prepared food, or "culture media." When thus cultivated it is found that different kinds of food often have a remarkable effect in modifying the form and reproductive activity of the fungus. Thus, the aquatic fungus *Saprolegnia mizta* tends to produce a vegetative mycelium only, without any spores, when supplied with abundant food regularly renewed. If such sterile, strongly growing cultures be transferred to plain water, or to a weaker food solution, sporangia with zoospores are formed in quantity. Transferred to a solid substance with little food, such as pure agar-agar jelly, sexual oospores are alone produced. Solutions of



leucoid or hæmoglobin are effective in stimulating the production of oogonia, and certain salts (phosphates, nitrates) have a similar action in regard to antheridia. Similarly the fungus *Pythium ultimum* forms only oospores when grown on cabbage leaves, whereas on other substances it bears conidia. Several species of the genus *Phytophthora* can be got to form oospores by appropriate feeding, though in some of them the sexual reproduction has never been observed under natural conditions. The mycelium of several common moulds (*Penicillium*, *Sterigmatocystis*, *Alternaria*) is quite different in structure with different food supplies; in some of the *Mucors* it may be changed wholly or in part from the filamentous to the gemmate condition. Still more remarkable is the production of distinct varieties of *Aspergillus niger* under the action of small doses of certain poisons. These "mutants" remain constant for many generations after the administration of the poison has ceased. It is possible that many cases of the succession of different kinds of spore in the one fungus are concerned with differences in the food available at corresponding periods of the life-history. For instance, the teleutospores of a rust may be formed instead of uredospores, because a particular sort of food, found in the plant at one time, may disappear, or a new sort may be provided.

### Symbiosis.

In all the cases of parasitism mentioned above, the fungus obtains its food at the expense of the attacked plant and gives nothing in return. The food-supplying cells are usually killed eventually by the excessive demands of the parasite. Some of the haustorium-forming species, as already said, are less exacting for the greater part of their lives, and only kill the host tissues when the production of spores makes a sudden exceptional call on their resources.

Several fungi are known which pass through their whole development at the expense of certain plants, but ordinarily cause them no injury. The Darnel grass (*Lolium temulentum*) generally contains an endophyte of this nature. So far as is known, this fungus gets the bulk of its food from the grass, but the latter does not suffer from its presence and even appears to be stimulated to more vigorous growth than when it is not there. It has been suggested that the endophyte assists in the supply of nitrogen to the host, by fixing the free nitrogen of the air.

In other cases, there is the clearest evidence that both fungus and other plant benefit from the presence of the former in the tissues of the

latter. The harmful effects of the fungus have been overcome and the attacked plant has been able to obtain some advantage from its presence. We apply to the life of these mutual benefit associations the term "symbiosis." It is doubtless probable that most of these associations arose, in the first instance, in an ordinary parasitic attack by the fungus; but to regard symbiosis in its present forms as a special case of parasitism, as is sometimes done, seems to be an undue stretching of the familiar meaning of the word "parasite."

The lichens form the most prominent case of symbiosis. As already stated they consist of a fungus and an alga living in association to form what appears as a perfectly distinct organism. A very large number of different "species" is known, but in all cases the arrangement is much the same. The fungus hyphæ surround and grow between the algal cells, sometimes sending haustoria into them, and feed in part on the organic food which the alga, by means of its green chlorophyll, is able to build up out of the carbon-dioxide of the air. The fungus, in return, appears to supply water, mineral food, and possibly peptone; it appears to have the best of matters, for it alone forms spores in most cases.

A very large number of plants form an association between certain of their roots and a fungus. The orchids are examples and also many forest and field plants, including a number of crops. These associations are known as "mycorrhizas" or fungus-roots. In many cases there is little doubt that the fungus began as a parasite, for indications of attempts on the part of the root to keep it out can often be found. Having once been penetrated by the fungus, the root cells seem to have successfully endeavoured to meet the attack and even to get something back in return for the food they give to the invader. In many roots, examination shows that in the outer layers the fungus has the upper hand and feeds largely on the starch stored up in the roots. In the inner layers the tables appear to be turned, for the fungus is gradually digested and appears to give up a considerable amount of its substance to the root. It is also probable that it helps the roots to obtain water. In orchids, the association is so close that the seed cannot be germinated in the absence of the fungus. In the common ling (*Calluna vulgaris*) also, the fungus is essential for full development, the seedlings remaining stunted and rootless in its absence. In most cases, however, there is no evidence that the mycorrhiza is necessary. One result of the measures taken by the root to protect itself, is that the fungi which form mycorrhizas appear rarely to produce

spores, getting usually only just enough food to enable them to live vegetatively; their cultivation in artificial media has also seldom been successful; and the species of fungi concerned are, therefore, not known, except in some orchids, *Calluna*, and some forest trees. Those so far determined belong to the *Agaricaceæ*, *Tuberaceæ*, *Sphæropsidaceæ*, and sterile forms allied to *Rhizoctonia*.

## CHAPTER III.

### LIFE-HISTORY OF PARASITIC FUNGI.

A PARASITE of the roots or stems of perennial plants or the leaves of evergreens has always available the food supplies requisite for its development. It may be, and often is, dependent on seasonal variations of activity, but is rarely put to such straits to tide over periods unfavourable to its existence as the forms which live on annuals or on the leaves of deciduous plants. Most of the common crop pests, and many other forms, have to face a period of the year when their ordinary supports are not available. In the plains of India, for instance, the wheat season is roughly from November to April, and it is exceedingly rare to find even a self-sown wheat plant between May and October. The fungi which are parasitic on wheat have, therefore, to pass through a period of about half the year in some other way than as active wheat parasites. The different methods adopted by fungi to solve this problem are of considerable interest, though they are not in all cases by any means completely understood.

In the wheat mildew, the greater part of the fungus dies off as the hot weather approaches. Late in spring, however, the perithecia of *Erysiphe graminis* are found, often in considerable abundance. Examined at this period they are seen to be immature and with the ascospores undeveloped. After a time, they may become detached from the mycelium and fall to the ground, or they reach the soil with fragments of the infected stubble. Here, internal development proceeds, and after some months, particularly if exposed to wet as during the monsoon, the asci mature and ripe ascospores are formed. These are ultimately set free by the rupture of the perithecium, and in the cold weather reach the growing wheat plants, no doubt usually with dust, and infect the crop. Many other Ascomycetes have fully-formed spores, while still attached to the host plant, but these spores, aided often by the protection afforded by the wall of the perithecium, preserve their power of germination for many months, even if exposed to considerable variations of temperature and humidity, as when cast into the soil. In deciduous perennials, the perithecia may lodge in

cracks in the bark and the spores be set free in the air in the following season. The flag smut of wheat, and many Peronosporaceæ, are carried over by the prolonged germinative capacity of their perfect spore forms ; and even the conidia of some imperfect fungi (e.g., *Alternaria Brassica* and species of *Fusarium*) and Ascomycetes have the same power.

In bunt, the spores (which are somewhat greasy, and can live for several years) remain adherent to the seed grain, which they reach during harvest, threshing, or storing, and are sown with the crop for the following season. Both parasite and host germinate together, and infection takes place in the seedling stage. Sometimes a second method is made use of. Under certain conditions, the bunt fungus can live as a saprophyte in the soil for a considerable time, budding off sporidia in large numbers. If this saprophytic life be prolonged, and the new wheat be sown in the same field, or manured with contaminated manure, infection may occur. Many other hemi-parasites and hemi-saprophytes owe their persistence largely to their power of passing a more or less prolonged period as saprophytes in the soil, or plant débris, until a new food crop is available.

The loose smut of wheat differs widely from the above cases in that it persists from one season to the next in the mycelial condition. As the crop ripens, the smut spores formed in the first-opened ears are blown throughout the field. Some of them fall on the feathery stigmas of recently-opened wheat flowers, germinate, and the germ-tube grows down to the ovary and infects the young grain. The mycelium remains in the grain in a dormant condition until it is sown, when it resumes activity and passes up into the seedlings, keeping pace with the growth of the latter until it is able to form new spores in the young ears. Other similar cases are known in several of the families of fungi. In the bean anthracnose, the beans become infected through the pod but, if the attack be mild, are not much injured and will germinate in the ordinary course ; the fungus grows into the young seedling and reproduces the disease in the new crop. The same occurs in gram blight. In potato blight, the fungus remains dormant in the tubers and, under certain conditions, will give rise to diseased shoots when the tubers sprout. Several other Peronosporaceæ also have the power of forming dormant mycelium in their hosts and, in some cases, this seems to be correlated with the absence of the sexually-produced resting spores, at least under natural conditions. Another interesting group of cases is that in which the normal " incubation period " (i.e., the time between infection and spore formation) is prolonged as a result of unfavourable conditions of temperature or nutrition

(see p. 45). The mycelium passes into a dormant state but is not killed, as its subsequent development proves (rusts of cereals and hollyhock). The term "hibernating" mycelium is sometimes applied to the mycelium in the above cases, but is evidently a misnomer if used in tropical countries, where the unfavourable season is usually the hot weather.

A very similar condition to the last is that known as "perennial" mycelium, where the parasite persists in the perennial parts of its host, usually in a dormant condition, and passes out into the new shoots or leaves arising from the infected part. One of the commonest of such cases in northern India is the rust of the weed often called "jungli gobi" (*Launea asplenifolia*). This plant has a perennial root stock, but the whole of the above-ground parts die down at the beginning of the hot weather and only appear again about October. The entire plant is often permeated by the mycelium of the fungus, which remains dormant in the roots during the summer months. Several other similar cases are known amongst rusts (e.g., on *Euphorbia*, *Anemone*), smuts (on *Scilla*, *Lychnis*), *Peronosporaceæ* (on *Helleborus*, *Ranunculus*, *Vicia sepium*), but none of these has been observed in India. Amongst species which are perennial in above-ground parts may be mentioned the peach leaf-curl fungus, common in the Himalaya. The parasite (*Ezoascus deformans*) has a perennial mycelium in the branches, which occasionally serves to infect the newly-opened leaves in the spring, though the majority of new infections come from spores which have remained alive on the bud scales and twigs from the previous season. In many of the "witches' brooms" (see p. 86) perennial mycelium also occurs. Amongst ectophytes, several mildews (*Erysiphaceæ*) are known, which persist in a dormant mycelial condition on perennial parts of the host, the perithecia being uncommon (mildews of apple and vine) or even unknown (*Euonymus* mildew).

Another type of persistence in the mycelial condition is found in many fungi which form sclerotia and other mycelial condensations. *Sclerotium Oryzæ*, for instance, probably lives between successive crops of rice chiefly as sclerotia in the soil.

In many cases, the parasite is capable of living on more than one host plant, sometimes on a large number belonging to several different natural orders (e.g., *Corticium salmonicolor*). This naturally helps to keep it alive, where suitable food plants are found throughout the year. The special variety of this condition known as "heterocicism" is more fully discussed below.

Finally, there are cases in which we do not yet know clearly how persistence of the parasite is effected. In the downy mildews of cereals (*Sclerospora*) attempts to carry over the disease from season to season by artificial means have failed; the special difficulties in the cereal rusts are referred to under those diseases; while in the tucourbit downy mildew the method made use of is entirely unknown.

### Heteroecism.

In some of the cases where the one species of parasite attacks two widely separated plants, the fungus assumes a different form on the two hosts, and passes one portion of its life in one form on the first plant attacked, and a second portion of its life in another form on the second. To this the term "heteroecism" is applied. Heteroecious fungi occur chiefly amongst the rusts, the only other known instance being the Ascomycete, *Sclerotinia heteroica*, a parasite which has not been found in India.

The best known case of heteroecism occurs in the black rust of wheat, *Puccinia graminis*. This destructive parasite passes the first part of its life on certain barberries, the second on wheat and other grasses. In the western Himalaya (as near Simla) the barberry rust is not uncommon, producing spermatogonia and aecidia on round patches, on the leaves, bright red above and paler below. The same fungus is known on barberries in many parts of the world. It never progresses (on barberry) beyond the aecidial stage, and as the aecidiospores have been proved incapable of infecting wheat, there is evidently something missing in its life-history. This gap was filled up when it was discovered that the spores were capable of infecting wheat and other grasses, and on these hosts caused the long-known black rust. The spores sown on wheat produced after a while a crop of uredospores, followed later on by the teleutospores. The latter were germinated and the resulting sporidia were shown to be unable to attack wheat, but to infect barberry leaves readily, giving rise to the spermatogonia and aecidia of the barberry rust. The cycle is therefore complete; two of the spore forms are borne on the barberry; two on the wheat or other grass; and one—the sporidia—is developed at the expense of the reserve food stores in the teleutospore.

In *Sclerotinia heteroica* the cycle is somewhat simpler. On one host, *Ledum palustre*, sclerotia are found in the fruit and develop, after a period of rest, into the apothecia of the ascigerous stage. The ascospores, however, cannot infect *Ledum* but, for their further development, must

reach the young shoots of a different plant, *Vaccinium uliginosum*. These they attack and produce a conidial form, very different from the ascigerous stage. The conidia again are unable to infect the host on which they develop, but if sown on the stigma of *Ledum* flowers, germinate, pass down to the ovary, and reproduce the sclerotia from which we started. There are only two spore forms, each on a different host.

Heteroecism in the rusts always follows a regular sequence; the æcidial stage is passed on the one host and the uredo-teleuto (or sometimes the teleuto only) on the other. It is probable that originally the parasite lived its whole life on one plant and later took to the habit of spending part of its life on a second. How this arose is, however, obscure.

If the heteroecious fungi were always obliged to go through their whole cycle of development, it is clear that they could only continue to exist so long as both host plants were available; it would then be possible to eliminate black rust of wheat, for instance, by destroying the barberry. Unfortunately this is not the case, for it has been found that the alternation of hosts is by no means essential to the life of the fungus. Black rust can persist in the uredo-teleuto stage for an apparently indefinite time. It has been prevalent in Australia for at least 90 years, though the æcidial stage has never been found, and there are no barberries; while in India the æcidial stage, found only in the Himalaya, cannot be connected with the common black rust of the Central Provinces wheat. Hence the significance, if any, of heteroecism remains unknown.

Outside the fungi, this peculiar habit of changing hosts is of still greater interest, owing to its connection with certain human diseases. Malaria, for instance, is due to a heteroecious animal parasite which passes one stage of its life in some mosquitoes and another in man.

### Specialisation of parasitism.

AMONGST those fungi which are capable of attacking several different species of plants, many have developed into distinct races, each of which, though outwardly similar to the others, is restricted to one, or a few only, of the host plants. A single species of fungus, such as *Puccinia graminis*, may include a number of these races, all quite similar in structure and not to be distinguished from one another in any other way than by their capacity for living on certain hosts. The difference between them is physiological not botanical, so that there may be several "physiological"



or "biological" species within the one botanical species. To this splitting of a parasite into specialised races, on different host plants, the term "specialisation of parasitism" is applied.

Every degree of transition may be found between those parasites which freely infect all their hosts (as *Pythium de Baryanum*), through those which attack other hosts less readily than the one from which they are taken (*Rhizoctonia*), to those that are narrowly specialised on a single host (*Erysiphe Polygoni*, on *Trifolium pratense*). Evidently the adaptability of the parasites to their hosts varies greatly in different fungi and is sometimes exceedingly close. Whole genera of fungi are known that are confined to a single natural order of plants, as the *Phragmidium* rusts are to the *Rosaceæ*. Still more frequently, a single species of fungus is restricted to a single species of host (as the castor rust), or to a single genus (as the cotton rust), or to a greater or lesser number of allied genera (as the potato blight fungus). There is nothing surprising, therefore, in finding a tendency amongst those fungi that attack many hosts to split into races, each specially adapted to life on a single one or a few of them. The splitting has even sometimes gone further, and within a single genus (or even species) of host plants there may be several specialised races, four or five being known in the brome grass mildew.

Specialisation of parasitism is strongly marked in many rusts and mildews (*Erysiphaceæ*). It is also found in the *Chytridiaceæ*, *Proto-mycetes*, *Exoascaceæ*, *Clavioceps*, and *Rhizoctonia*. In the *Peronosporaceæ* it occurs, but not very strongly marked, in *Cystopus candidus*, and is probable in *Sclerospora graminicola*. A few smuts are said to have specialised races (*Ustilago violacea*). In the common bean anthracnose (*Glomerella Lindemuthianum*) there is evidence of distinct races occurring on different varieties of *Phaseolus vulgaris*, but the allied *Glomerella cingulata* shows little sign of specialisation. *Epichloe* and *Sclerotinia* are amongst the other *Ascomycetes* believed to have this character and probably many more will be added in future. It is clear that the relative immunity to the attack of certain parasites, displayed by some of the varieties and races of cultivated plants, is often only a consequence of specialisation in the parasite and this aspect of the matter will be returned to later on.

Specialisation has been very fully investigated in the rusts and mildew of the commoner cereals. Wheat, barley, and oats have each their distinct races of rust and mildew. The black rust of wheat will not attack oats in India but sometimes passes to barley. That from barley

infects neither wheat nor oats. On oats, black rust has not hitherto been seen in India, but in other countries it rarely passes to wheat or barley. In Europe, the black rust of wheat may attack oats directly, and in the United States will do so sometimes, if grown first on barley. Hence the specialisation of a given parasite may vary in different localities, several such cases being known.

The cereal mildew (*Erysiphe graminis*) has still more strongly specialised races. The form on wheat will ordinarily only infect species of *Triticum*\*; that on barley, species of *Hordeum*; and that on oats, species of *Avena*. Not all the species are equally attacked, however, as some species of *Hordeum* resist barley mildew. Even within a single species, the varieties may differ in their resistance, as in emmer wheat (*Triticum dicoccum*), in which, of 5 varieties tested in one case, 2 were quite susceptible, 2 resistant, and 1 immune. Emmer behaves in much the same way with regard to rust.

As a general rule, the infective powers of a specialised race are identical in all its spore forms. Thus, in black rust, the æcidial form on the barberry has the same specialisation as the uredo-teleuto, its spores being only able to infect the same host plants as bear the teleutospores from which it originated. An æcidium derived from infection by sporidia of oat rust will not serve to transmit the rust to wheat or barley. In *Erysiphe graminis* also, the conidia and ascospores have exactly the same powers, and this is true likewise for the five races known in *Claviceps purpurea* (ergot). The rule is not, however, absolute, for some varieties of asparagus which resist attack by the uredo stage of the asparagus rust, succumb to the æcidiospores.

Specialisation is a character which can be considerably modified by changes affecting either the host or the parasite. In its origin at least, it was clearly an "adaptive" character, and as such has preserved a certain measure of plasticity. Amongst the changes in the host which alter its resistance to a race of the parasite to which it is normally immune, are several that seem to act by lowering the vitality of its cells. Thus, barley leaves can be rendered liable to attack by *Erysiphe graminis* from wheat in several ways. Touching them with a hot knife makes the spot touched susceptible. Cutting off some of the cells (and thus injuring the adjoining ones) has the same effect. Exposing the leaf to the

---

\* There is an exception to this in the grass known as *Hordeum silvaticum*, young plants of which are attacked by the wheat mildew while resistant to that from barley.

vapour of ether, chloroform, or alcohol makes it liable to attack in any part. Since the fungus is a true parasite, incapable of growing on dead matter, it is necessary to graduate the injuries so as to lower vitality without actually killing the cells. Such injuries are not uncommon in nature, where they produce just the same effect. For instance, a leaf partly eaten by slugs becomes susceptible near the edges of the bitten places. Frost, hail, and the like may act in the same way, and also apparently green-fly and other insects that suck the juices of the plant. Even the agricultural operation of rolling the crop may destroy the resistance of the bruised leaves. High fertilisation has also been found to increase the susceptibility of cereals to races of black rust to which they are normally semi-immune, as wheat and rye to the race on oats.

In all these cases, the immunity of the host is modified without the parasite becoming altered in any way. The spores borne on the injured parts remain incapable of infecting the sound tissues in the neighbourhood. It has not hitherto been found possible to "educate" a true parasite by any artificial method into attacking the perfectly sound tissues of a host that is normally quite immune to it, in the same way that, as already seen (p. 43), saprophytes may sometimes be trained into the parasitic life. Nevertheless, it is known that the virulence of those more weakly specialised parasites that can attack several hosts, but some less readily than others, can be modified by being confined to one host for a number of generations. Thus, einkorn wheat is relatively resistant to black rust, that is to say it becomes infected, but the attack is very mild. If, however, the fungus is kept growing on einkorn for a number of generations, it gradually increases in virulence until fairly severe attacks may be obtained. In the same way, *Rhizoctonia* from carrot can grow on beet, and after a few generations on the latter host becomes distinctly more virulent and has a greater disorganising action than at first. Furthermore, there is at least one circumstance in nature which is capable of so modifying the internal disposition of a true parasite as to render it capable of attacking a host normally quite immune to it; this is growth for a time on some third species of host plant. It has already been mentioned that, in the United States, black rust of wheat will sometimes infect oats, if first grown on barley (on which it is habitually capable of growth), though direct infection of oats from wheat does not normally occur. The term "bridging species" is applied to the barley in this case. Very few instances are known of the occurrence of bridging species. Rust and mildew on brome grass have them, so has black rust on wheat, and there are one or two other cases. Perhaps the most interesting recorded is that with black

rust, in which the hybrid produced by crossing an immune with a susceptible variety of wheat served as a bridge to the immune parent. The first generation of the cross was very badly infected, and spores from it readily attacked the previously immune parent.

It appears that two or more distinct physiological species may live on the one host plant. This occurs in black rust in the United States and possibly also in bean anthracnose. Whether the difference in the specialisation of a parasite in different localities is due to the presence of quite separate races in the two areas, does not seem to have been tested experimentally, but it is an obvious possibility.

### Infection.

In order to obtain their food, parasites have to establish a close connection with their host plants; as a rule, this implies that they have to enter into the tissues or to send haustoria into the surface cells, but in the pure ectophytes close union with the outer surface, with possibly some chemical alteration of the membrane, seems to be sufficient. To this establishment of parasitic relations the term "infection" is applied. In some cases, two distinct stages may be recognised, a stage of "penetration" preceding the stage of infection proper. This has been observed in some endophytes that enter by the stomata (as the rusts), in a few subcuticular parasites (*Venturia*), and in some haustorial ectophytes (*Erysiphe graminis*); and in such cases penetration is not necessarily followed by the establishment of the parasitic life.

Penetration may take place in uninjured green parts through the stomata, or by direct passage through the outer wall of the epidermis. In woody or corky parts, it may occur through the air passages of the lenticels, through cracks in the bark, or directly through the cell walls.

Entry appears to be induced by certain physical and chemical conditions of the host or of the environment. In the rusts, it is said to be probably due to the greater moisture of the air in the cavity of the stomata ("hydrotropism"), or sometimes to growth away from light ("negative phototropism"). In other cases, chemical attraction ("chemotropism") has been said to be the cause. It has been stated that certain chemical substances exercise an attractive influence on the tips of germ-tubes and hyphæ. These include bodies containing nitrogen, phosphates (meat extract), and carbohydrates (sugar), allies of which are normally found in the cells of plants. Acids, alkalis, and certain salts, on the other hand, repel the hyphæ. Hyphæ of non-parasitic fungi have

been induced to penetrate living plants injected with meat extract or sugar (*Penicillium glaucum*), and parasites to enter hosts on which normally they cannot grow, when injected with an attractive substance such as an extract of the leaves of the normal host (wheat rust on *Tradescantia*). Other observers have failed to confirm these results, so that it is doubtful what part, if any, chemotropism plays in infection. It has not been shown, also, in most of these cases, that the fungus can continue to grow as a parasite inside its new host. Indeed, in the rusts, it has been found that the germ-tubes of the uredospores will enter the stomata of wrong hosts readily enough, but soon die, except when sown on hosts on which they are normally found; the first stage, that of penetration, is alone secured, but true infection is not obtained.

The most primitive group of parasitic fungi is the Chytridiaceæ. In these, the zoospore comes to rest on the surface of its host, surrounds itself with a wall and then, after a short period of rest, puts out an extraordinarily slender tubular process (often not a micron\* in diameter) which penetrates through the cell wall into the interior of the host cell (Fig. 17, 1). Through this tube, the contents of the spore pass, leaving the empty spore-shell behind. In a few cases (the parasite of "brûlure" of flax and that of warty disease of potatoes) the whole spore is said to make its way bodily through the cell wall, as also occurs in some myxomycetous parasites.

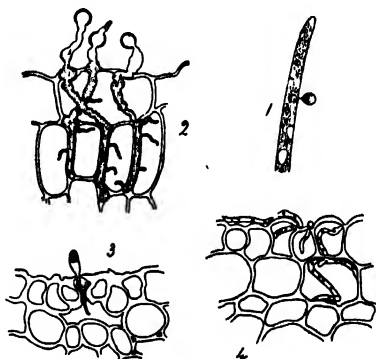


FIG. 17. 1, zoospore of *Pleoiphidium irregulare* infecting hypha of *Pythium*,  $\times 500$ ; 2, zoospores of *Phytophthora Colocasiae* infecting leaf of *Colocasia*,  $\times 350$ ; 3, conidium of *Enobasidium vesans* infecting leaf of tea through stoma,  $\times 400$ ; 4, hyphae or germ-tubes of *Colletotrichum falcatum* infecting root of sugarcane,  $\times 250$ .

The zoospores of several Peronosporaceæ and the sporidia of the rusts behave in a similar manner to the Chytridiaceæ, except that the

\* The *Micron* ( $\mu$ ) is the unit of microscopical measurement. It is the one-thousandth part of a millimeter or the one twenty-five-thousandth part of an inch.

tube of entry is larger and may sometimes grow as a germ-tube for a little distance on the surface of the host, before penetrating the wall. In these cases, the parasite is not usually found in the interior of any cell but that by which it has entered the host, the subsequent life being intercellular as a rule, except for haustoria (Fig. 17, 2).

In other cases, a superficial mycelium of greater or lesser extent may be formed before penetration occurs. In *Leptosphaeria Tritici*, for instance, the germ-tube branches and each of the hyphae may penetrate by its extremity directly into a cell of the host. In "pink disease" of rubber also, the mycelium is at first entirely superficial and penetration only occurs later.

Occasionally (*Ustilago Zeæ* on maize), the germ-tubes (promycelia) from the primary spores of smuts do not seem capable of infecting, only the sporidia borne on them being able to give a parasitic germ-tube. The promycelia of the rusts seem also to be ordinarily saprophytic, but their sporidia are capable of causing infection.

In some cases (even in such a definite parasite as oat smut) penetration of perfectly sound external cells does not occur. The cells must be languishing or moribund before they can be penetrated. In oat smut, the mycelium becomes intercellular during the subsequent growth in sound tissues. There are many cases where penetration is assisted by weakening of the epidermal barrier, though once this is crossed, growth into healthy tissues may occur freely. A few parasites of leaves and fruit are assisted in their penetration by the presence of foreign matter on the surface. *Botrytis cinerea* has been found to attack sound leaves of dahlia and pelargonium on which fallen flowers were decomposing, and *Monilia fructigena* sound fruit soiled by juice from other fruit.

Penetration through the stomata occurs in the æoidiospores and uredospores of rusts (Fig. 102, 4-5), in some Phycomycetes (e.g., *Cystopus candidus*) and Basidiomycetes (e.g., *Exobasidium vexans* (Fig. 17, 3), and in many Ascomycetes and imperfect fungi (Fig. 141). Sometimes penetration may be both through stomata and directly into epidermal cells (*Peronospora parasitica*). After entry into the sub-stomatal space, the hypha may swell up into a vesicle, from which one or more branches arise and grow towards a neighbouring cell into which a haustorium is sent; infection is not complete until this occurs (rusts, *Plasmopara viticola*). In other cases, the infection hypha proceeds directly to invade the deeper tissues, branching as it goes and passing either

between the cells or through them (banded sclerotial disease of sugarcane).

The penetration of woody or corky parts is most frequently effected through breaks in the bark, whether due to the formation of lenticels, or to wounds, or natural cracking of the outer layers. Entry through lenticels has been reported in the European canker fungus, *Nectria galligena*, though this is usually a wound parasite, and in *Rhizoctonia destruens* on potato tubers in India. Potato tubers can be infected by *Phytophthora* through the eyes, lenticels, or wounds. The red rust of tea enters through cracks in the bark, and the susceptible period is usually confined to the time during which the twigs are covered with scaly bark. The majority of the parasites of woody parts, however, enter through wounds or injuries to the outer cell layers. Most of the stem parasites of trees infect through wounds, especially those due to the breaking of branches or the action of insects. In the mulberry disease in Kashmir, caused by *Coryneum Mori*, the entry is both through wounds formed by breaking off the branches, and at points injured by frost and snow, near ground level and in the forks of the branches. *Nectria cinnabarina* is said to attack the mulberry in Europe through buds injured by frost. In these cases, the hyphæ grow into the dead cells near the surface, and from them extend into the sound tissues further in.

In some cases, stem parasites can enter through dead branches, as in the "die back" disease of rubber, where entry has been observed to occur through the lower branches that die naturally as the tree grows, and also through branches infected with *Gloeosporium*, or *Phytophthora*, or with pink disease. In many of the parasites that enter through injured or dead parts, the fungus begins as a saprophyte, and only becomes parasitic after having accumulated a certain amount of energy by feeding on dead cells.

Infection of roots may be by direct boring through the uninjured epidermal or cortical cells (*Colletotrichum falcatum* (Fig. 17, 4), *Fusarium wilt*) or through wounds or cracks in the bark (*Thyridaria tarda*, *Fomes annosus*). Usually, the first type occurs in young roots, the second in older ones. Sometimes (*Ustilina zonata*), a fungus can penetrate the sound roots of seedlings, but on older plants enters through wounds. This is doubtless due to the protection given by the corky outer cells of the older roots.

In some of the above cases, infection is caused directly from the spore, the germ-tube of which enters before the reserve food supply, which it

obtains from the stores accumulated in the spore, has been exhausted. In others, a mycelium of greater or less extent is first formed. Indeed, in some cases (some Rhizoctonias and other sclerotial forms), spores are not known, and the whole life-history seems normally to be passed in the mycelial condition. There is no essential difference in the process in the two cases, though it appears probable that the enzymic activity, to which penetration is often due, is greater in the young germ-tube than in the older mycelium (*Botrytis cinerea*).

Many parasites can only infect through definite parts of the host plant, though the subsequent growth may be diffused, and spore-production may occur at a distance from the point of entry. In oat smut, for instance, infection occurs through the mesocotyl of the germinating seed, but the spores are formed some months later in the ears. In the loose smuts of wheat and barley, and in some Sclerotinias, infection occurs through the stigma of the flower; in ergot, through the young ovary; in maize, through any young dividing tissues, and so on. In the Chytridiaceæ, localisation is sometimes very strongly marked. Some enter and complete their development in a single hair, whether of the leaf (*Synchytrium trichophilum*) or of the root (*Asterocystis radialis*), this latter is further limited to the short growth-region of the root. Age of the tissues is often an important factor, penetration being only possible in many cases through young parts (sporidia of rusts); but the different spore forms of a fungus do not necessarily agree in their infective powers (the æcidiospores and uredospores of the rusts can penetrate mature leaves). The thickness and composition of the outer cell walls is the essential feature in some of these cases, and in many plants there are permanent weak spots in the defences, as at the nodes in sugarcane and just above the nodes in rice.

In a few cases, parasites are known which only grow on the abnormal tissues produced by certain gall-forming insects or mites (*Sphærothea phytoptophila* on mite galls of *Celtis*). It is not certain, however, whether this is due to entry being possible only in such localities, or to the subsequent parasitic life of the mycelium being checked when penetration takes place elsewhere.

Prior to penetration, some parasites form special organs to which the name "appressoria" has been given. The precise functions of these organs are not known, but they are probably concerned with an accumulation of enzymic energy. They are most commonly found in the mildews, rusts and anthracnoses, but occur also in a few other cases.



In the mildews, they are simple or lobed outgrowths, usually from the side of a hypha, formed in close contact with the surface on which it is growing (Fig. 18, 1). In those species in which they occur, the haustoria arise only from them or from the hypha at the point where they originate. In *Erysiphe Polygoni*, and probably in all the others, they will form in contact with any hard substance, but only give out an infection tube when on the epidermis of the host plant. In the rusts, they are less specialised, being simple swellings on the tips of the germ-tubes, which develop as soon as the mouth of a stoma is reached (Fig. 18, 2-4). An infection tube arises from the lower side, passes through the opening of the stoma, and swells up below into a vesicle into which all the contents of the germ-tube pass. From this vesicle, the true infection hyphae arise.

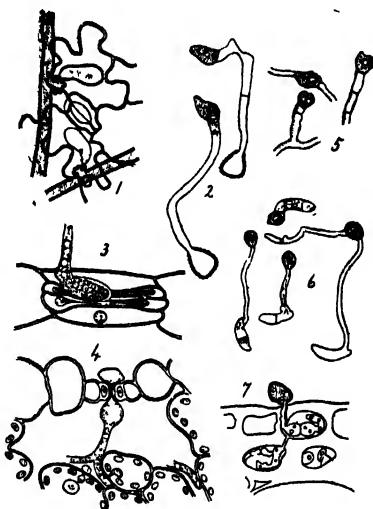


FIG. 18. Appressoria: 1, of *Erysiphe Polygoni*, with haustoria,  $\times 300$ ; 2, of *Uredo echinulatus*,  $\times 125$ ; 3, of uredo stage of *Puccinia dispersa*; 4, substomatal vesicle and infection hypha of *Puccinia triticea* (uredo); 5, appressoria of *Colletotrichum falcatum* on hyphae of the mycelium,  $\times 250$ ; 6, appressoria of *Colletotrichum falcatum* on germ-tubes of the conidia, one has germinated,  $\times 250$ ; 7, infection of sugarcane leaf by a hypha from one of the same,  $\times 250$ . (1 after de Bary, 3-4 after Pole Evans)

Appressorial formation is not a necessary preliminary to infection (as they sometimes fail to form on the penetrating germ-tubes of the uredospores of *Hemileia vastatrix*); and they may be produced and give an entry tube on wrong hosts. In the anthracnoses, they are still less specialised, as they act also as chlamydospores, being able to give out a germ-tube and form a new mycelium even when not on the living plant (Fig. 18, 5-7). Contact with a hard substance is also not necessary (though it stimulates their production) for they may be found in quantities throughout crowded or old cultures. In all these cases, the appressoria are unicellular, but multicellular appressoria are also known

(*Cercospora vaginata* on sugarcane). In some cases, the appressorium has a further function as an organ of adhesion to the surface (*Sclerotinia*, *Venturia*).

The conditions influencing infection are numerous and involved. In the first place, there are those which enable the parasite to reach its host. They have been discussed in the section on dissemination (p. 32) and it only remains to add that the chances in favour of the fungus finding its billet may be greatly modified according to the system of cultivation adopted in particular crops. It is obvious that they are greatly increased when large areas are continuously covered with the same crop, as in tea gardens, vineyards, and the like. Not only is the host easily accessible, but the quantity of infective material is increased. It has been calculated that vine mildew spores may fall at the rate of 2,000 per square inch in 24 hours, in badly infected areas in France. Thread blight in tea would be of trifling importance if the bushes were isolated.

Then there are factors influencing the germination of the spores, and others which affect the receptivity of the host.

Moisture is probably the most important condition which controls the germination of the spores. Most spores require moist air for their germination and some must have free liquid, as rain drops or dew films (many *Phycomycetes*). The spores of the common scab fungi of apples and pears (*Venturia*) will not germinate unless completely immersed in water. It is a matter of simple observation to note how much more frequent are, for example, leaf-spotting fungi in the moister parts of India than in the dry north-west, and in dense jungle than in open fields. On some coffee estates it is possible to predict where koleroga will be severe by noting where the mist hangs in the monsoon months. Pink disease often begins in the forks of rubber trees, because of the accumulation of water there. Soil moisture influences notably the germination of such spores as those of the smuts that are shed in the ground, and this may have an important bearing on their persistence from season to season (flag smut of wheat). Complete immersion of the spores may check infection in some cases; the sporidia of rusts are not formed so long as the promycelium is wholly immersed, while the appressoria of *Hemileia vastatrix* usually only appear as the surface of the coffee leaf dries up. Prolonged or excessive moisture will also check the infection of clover leaves by ascospores of *Sclerotinia Trifoliorum*; the appressoria are not formed if a film of liquid prevents close contact with the leaf surface,

and the enzyme necessary for penetration is possibly not produced when the germ-tube is stimulated to too vigorous growth by the presence of plenty of water, or, if formed, may become too diluted in the liquid. Alternate damp and dry periods are best in these cases. A preliminary drying of the spores is sometimes an advantage (uredospores); this may act by maturing the spores, as it is probable that water is lost during the process of ripening. Freezing, another way of drying out superfluous water, has the same effect.

Temperature is often but little less important. There is an optimum temperature, at which germination is most free; and maximum and minimum temperatures, above and below which germination fails. The potato blight fungus will not develop in the hot weather in India; its optimum for spore germination is about 50° to 55°F., and at this temperature infection occurs most readily, not because the host plant is more susceptible, but merely because the spores germinate with greater rapidity and more abundantly than at higher or lower temperatures. In a number of cases, chilling the spores increases germination (ooidia of Erysiphaceæ, uredospores of some rusts, smut spores, sporangia of several Phycomycetes). Artificial infections carried out with *Cystopus candidus* gave 95 per cent. of successes when the spores were chilled to about 50°F. (the optimum for germination), while at temperatures near 75°F. few succeeded. In this fungus, the maximum for germination seems to be about 77°F. and the minimum near freezing point. On the other hand several common moulds (*Aspergillus*) have their optimum in the neighbourhood of 90°F., bunt of wheat above 60°F., and so on. In several Phycomycetes, it has been observed that infection takes place most readily in the early morning, when not only is the temperature at its lowest, but films of dew are freely deposited.

Light sometimes has a considerable effect. Cultures of *Phytophthora parasitica* kept in the dark tend to remain sterile, and darkened sporangia fail to form zoospores. This does not occur in the allied *Phytophthora Colocasiæ*. The uredospores of rusts may fail to infect plants in darkness. In *Hemileia vastatrix*, germination is best after a short exposure to strong light, but a long exposure hinders it. Only the blue end of the spectrum is effective in this case, the red being inert. An exposure of previously darkened spores for 30 minutes to the blue rays gave 27 to 36 per cent. germination, while if exposed for an hour only 3 to 6 per cent. germinated, and if unlighted 9 to 17 per cent.

Of the factors influencing the receptivity of the host, age, the condition of the outer walls, and the nature of the cell contents, are amongst the most important. In young parts, the cuticle is usually less resistant, but, on the other hand, very young parts may be thickly clothed with hairs, or have a continuous epidermis which later disappears. Several parasites that penetrate only through young parts have already been mentioned. In black rot of the vine, however, the very young leaves escape, no doubt in part owing to their dense covering of hairs; the same is probably the reason why the hairy lower surface of apple leaves is seldom attacked by *Venturia inæqualis*, the most serious parasite of the crop in Kashmir. Young beet leaves, too, are ordinarily immune to leaf spot (*Cercospora beticola*) because their stomata do not open sufficiently to allow the germ-tubes to penetrate. The intact epidermis of young shoots of tea is not usually penetrated by the red rust alga, but entry occurs when the early formation of bark causes the surface to crack. The actual thickness of the cuticle may be modified, even in adult parts, by shade and humidity, and also often differs according to the variety of the host. Germ-tubes that penetrate through the cuticle (as the sporidia of the rusts) have less difficulty in crossing the thin walls found in densely shaded or overcrowded plants, while the higher humidity of the air under these conditions also assists germination. In the same way, the thin cuticle of certain varieties of grapes and apples makes them more susceptible to the attacks of *Botrytis* and *Venturia*. The waxy covering of some leaves and fruit, which varies in different varieties, may also play a similar part (white rot of grapes); or may act merely by preventing wetting of the leaves and allowing the spores to be easily washed off (barley rust). In stems and roots, the formation and thickness of the corky layer often influences penetration (*Rosellinia*, *Ustilina*). In *Venturia pirina* and *Corticium salmonicolor*, the shaded side of branches, where cork-formation is late and the cuticle thin, is most affected. The tough rind of the sugarcane is impenetrable by the hyphæ of *Colletotrichum falcatum*, though infection through the eyes and dormant adventitious roots at the nodes occurs readily. All these barriers may naturally be removed by wounds, and the large group of wound parasites are dependent on this removal for their infection. Finally, those parasites that enter by stomata are restricted to areas provided with these organs, as, for instance, *Hemileia vastatrix* to the under surface of coffee leaves.

Of the factors depending on the nature of the cell contents which influence the receptivity of the host, it is difficult to separate those that

affect penetration, from those that assist or hinder the establishment of the parasitic life; this will be referred to again under "Predisposition and Immunity" (p. 121). It has already been mentioned that rusts can penetrate unsuitable hosts but soon die, while *Erysiphe graminis* from wheat will form haustoria on barley, but these again degenerate in a few days. Even where a high percentage of successful infections is obtained, the host may be seen to be uncongenial by the slow subsequent development and lesser production of spores (cereal black rust on resistant varieties of the host). Many of the numerous "physiological" factors which have been observed to increase susceptibility to attack, probably act rather by assisting subsequent development than by making penetration easier. In others, even this action is absent and the increased damage is due to weakness of the host, and not to greater susceptibility to the parasite. Thus it has been observed that heavy bearing increases the damage to coffee from leaf disease, and heavy plucking that to tea from grey blight, though the fungi that cause these diseases are probably not influenced in their penetration or subsequent growth by any such conditions. Indeed it is well known that coffee leaf disease appeared first on some of the best coffee in Ceylon, and that it may take a number of years for it to weaken the bush seriously. It has been established in this case that infection and growth occur better on strong leaves, rich in nutriment, than on those with less food supply, but whereas impoverished plants soon show the effect of the drain on their scanty resources, vigorously growing bushes hide their losses, until repeated attacks turn the balance against them.

In any case, we should distinguish between infection and the subsequent growth of the parasite, and this is often difficult, or at least exact observations are scanty. It is known in some cases that artificial inoculation fails unless the host plant is healthy (*Cystopus candidus*, *Erysiphe Cichoracearum*), in others unless the host plant is feeble (weak parasites such as *Cladosporium herbarum*); but whether this is due to some innate (physiological) condition of predisposition (see below, p. 120), which helps the parasite to live and grow in its new surroundings, or to actual penetration being facilitated is, in most cases, obscure.

At the same time, if the experiments mentioned above (p. 64), on the influence of certain substances in attracting or repelling the tips of the hyphae, are of general application, it is probable that actual penetration, as distinct from subsequent true infection, may be influenced by conditions that alter the nature of the cell sap, such as nutritional

changes, manuring and the like. It is better, however, to defer the further consideration of these factors to the section on "Predisposition and Immunity."

### **Growth and reproduction of parasites.**

The subsequent growth after infection does not offer any special points of interest, other than those concerned with the arrangements for coming into contact with, and absorbing the food, already discussed, and those which result from the alterations in, and reaction of the host plant, which will be separately dealt with. The reproduction also has no special features beyond those already described.

---

## CHAPTER IV.

### THE CAUSATION OF DISEASE BY FUNGI.

DISEASE in plants may be defined as variation from normal physiological activity, which is sufficiently permanent or extensive to check the performance of the plant's natural functions or the completion of its development. In health, a plant is in what might be described as a condition of equilibrium, that is to say, all its internal functions and processes are graded to suit a particular set of external conditions. Its roots take up the right amount of water to keep the tissues well supplied, and this water contains the right amount of mineral food dissolved in it; its leaves manufacture, with the aid of the sun, enough starch to supply its requirements, and so on.

Anything that causes a plant to depart permanently or seriously from this condition of equilibrium produces disease. Thus if the roots are in soil so dry that they cannot obtain enough water, the leaves wither and the whole plant eventually dies; while if the plant be kept in the dark, it cannot form enough chlorophyll and starch, and becomes drawn, bleached, and feeble.

The attack of a parasitic fungus may produce similar effects. A plant may be growing in soil sufficiently moist to supply its water requirements, but the attack of a parasite may kill the root cells, or plug the water-carrying vessels. The result is a "wilt disease," the plant withering just as in a prolonged drought. Or a fungus may attack the leaves and destroy so many of the starch-manufacturing cells that there is not enough left to enable it to complete its development, and it remains stunted and unable to mature its fruit. These are only two of the many ways in which a fungus may injure the health of a plant: some manufacture poisons which kill the cells; others consume the food which should go to feed the plant; others prevent it forming its seeds by destroying the flower or fruit; others set up rotting of some vital part such as the base of the stem, so that the above-ground part collapses or is cut off from the roots. No part of the plant is secure from attack, and there is scarcely one of its functions that cannot be injuriously affected.

Many parasitic fungi are local in their attacks. Some are found only on leaves, some on stems, others on roots; these are instances of localisation on particular parts or organs of the host. There may be other kinds of localisation, as when each point of infection leads only to a small patch of disease but the patches occur scattered over the plant (as cereal rusts on all the green parts). Again there may be localisation to particular tissues, though any part of the plant which contains such tissue may be infected (e.g., maize smut on any embryonic tissue).

Other parasites are general and may spread through all, or a great part of the plant (e.g., *Phytophthora infestans*).

Even the local parasites may cause death, either by attacking a vital part or by multiple injury. The bud rot of palms in India is confined to the crown of the tree, but is usually fatal since it ultimately reaches and kills the single growing point at the top of the axis. Groundnuts may be killed by *Cercospora personata*, owing to the excessive loss of leaf caused by this fungus. General parasites are very often rapidly fatal owing to their power of causing very extensive injury in a short time.

It often happens, particularly with local attacks, that the injury caused to the general health of the plant is slight. There is naturally every gradation between, say, a leaf-spotting fungus which causes a minute spot here and there on the leaves but has no appreciable effect on the health of the plant, and such a fulminant attack as that of *Pythium de Baryanum* or *Phytophthora infestans*, which may bring down the whole plant, in twenty-four hours, in a rotting mass.

The injurious effect of a parasitic attack may be long delayed. The remarkable case of the cereal smuts, where the fungus grows up from the seed within its host plant, but causes no apparent injury until the ears begin to form, has already been referred to. In other cases, the damage from the first attack is slight, but the action is cumulative and the plant may ultimately succumb to repeated attacks. This is particularly the case with certain parasites that infect leaves or young twigs. The plant can often resist the loss of a certain number of leaves and twigs, replacing them by the development of others. When these in turn are attacked before they have had time to restore the normal equilibrium of the host and replenish the food reserves drawn on in their production, the net result is a slight reduction in the resisting powers of the host. Recuperation becomes a little more difficult each time, and may ultimately fail and the plant dies. This succession of events can



be observed in perennial crops, such as tea or coffee attacked with red rust or leaf disease. It is also very evident in the common "wither-tip" of orange trees in India.

Up to the middle of the last century there were still botanists of repute who maintained that parasitic fungi were only products of diseased tissues and not independent organisms; they were regarded as effects and not causes of disease. In the next twenty years, however, it was clearly established that these parasites were separate plants, and that by applying their spores to perfectly sound individuals of their hosts, disease could be artificially produced. Later, it was found that many of the parasites could be cultivated separately on prepared food, and a ready and convenient method of proving that they caused specific diseases became available. With certain precautions to avoid contamination, it is possible to grow many parasites on various organic juices, soups, jelly, and the like, in closed flasks or other vessels. A "pure culture" may be obtained, consisting of only a single organism, and by inoculating a healthy plant with a part of the growth, the disease which the parasite is capable of causing can be reproduced. Where the parasite has not been cultivated independently on artificially prepared substances (as in "true parasites" like the rusts), the disease has been shown to be produced when one or a few spores are sown on the host plant. The majority of our commoner crop diseases have thus been successfully reproduced by artificial inoculation.

In the study of any disease, it is necessary to determine its nature ("Diagnosis"), to study its cause ("Ætiology"), its effects on the tissues or structure of the host ("Morbid Anatomy"), in order to devise rational measures for its treatment ("Therapeutics").

### Diagnosis : Symptoms.

The diagnosis of a disease means the discovery of its identity or nature. Symptoms are the outward signs by which a diagnosis is assisted.

When plants are seen withering in the fields in time of drought, past experience tells us that this is due to want of water at the roots. But exactly the same kind of withering is common in many parts of India in fields of pigeon pea or cotton, even when there is plenty of moisture in the soil. Experiments have proved that these cases are caused by the attack of a fungus parasite (*Fusarium*) at the roots and base of the stem. Hence the same symptom may indicate any one of several causes,

and diagnosis consists in ascertaining the particular cause at work in each instance.

The methods of diagnosis employed in phytopathology have one great advantage over those used in the study of human diseases. It is permissible to cut up a living diseased plant to look for an internal parasite, and having found one, to endeavour to reproduce the disease by inoculating a healthy plant of the same kind with the suspected parasite. This is rarely possible with human beings. Hence it is that the study of the outward effects caused by disease in plants has not been carried to the same perfection as in man. The direct experimental method of finding the parasite and using it to reproduce the disease is so much more certain in its operation, that the specialised powers of the fine diagnostician are rarely required. It may be also that the symptoms of disease in plants are more general and difficult to differentiate than in animals, owing to their organisation being less specialised. A direct diagnosis by the observation of symptoms alone can, however, occasionally be made.



FIG. 19. Withering of orange from foot rot (late stage).

The following are some of the symptoms most commonly observed; they are at times characteristic enough to furnish exact information; but, as will be seen, there is often considerable risk of mistake unless observation is checked by experiment.

**WITHERING and FALLOR.**—The first and clearest indication of a fungus attack is often the withering of a whole plant or of some part of it.

The term "wilting" is sometimes applied to those cases where a whole plant dries up more or less suddenly from an attack at the roots or base of the stem. It may be confounded with the effects of drought or frost, but may usually be distinguished by the absence of these conditions and by its appearance in isolated plants or patches in affected fields. Some insects (e.g., cotton weevil) cause similar symptoms.

Drying up of a branch occurs sometimes in early stages of wilt (pigeon pea). More usually it is due to the infection of the branch by one of the canker fungi, or, in woody plants, by a wound parasite. The particular form known as "die back," where twigs dry up from the tip downwards, is sometimes due to infection by one of the anthracoses. Loss of leaf, such as occurs in the coffee leaf disease, may also lead to the death of branches or twigs. A similar appearance may be caused by the action of frost on young wood, by severe insect attack, and by any excessive defoliation. The "stag headed" condition of full-grown trees is due to the tree being unable to form leaves on its topmost branches and to the latter drying up, as a result of excessive drainage or impoverishment of the soil.

Pallor is due to destruction of chlorophyll or the checking of its formation in leaves. It is a common sign of the presence of a parasite in or on the pale area, but is also produced by the non-parasitic sooty moulds, which cut off light from the parts below. Sometimes it is caused by a stem attack lower down, all the leaves above the point infected being discoloured. An extreme case of this is found in the stem form of red rust on tea, the leaves being turned white. Pallor is also due to water-logging, to over-crowding, deficiency of iron in the soil, low temperature and other conditions, which can often be distinguished only with difficulty.

**DAMPING OFF.**—This name is applied to the sudden collapse of seedlings, which are attacked at the base of the stem and fall over from weakening of the tissues at this point. It results from the rot caused by several fungi, as *Pythium de Baryanum* and *Rhizoctonia*, and is common in other countries in gardens but is not often seen in India. A similar condition is sometimes seen in Bihar in crops such as indigo and jute, when a light rain falls as the seedlings are coming up; this seems to be non-parasitic.

**LEAF-SPOTTING.**—Discoloured spots on leaves form one of the commonest signs of the presence of a leaf parasite. These spots are very varied in colour, according to the plant and parasite concerned, and

also often change colour at different stages in their development. In some cases, the colour is not uniform, but zones or bands of different shades may alternate (Fig. 124).

Pallid spots have been referred to above. They are common in the mildews and in early stages of many other parasitic attacks. Insect punctures are often hard to distinguish, except by microscopic examination.

Yellow spots are caused by rusts and some other fungi. Very often the colour changes to brown or black at a later stage. They are often raised and covered when mature with a yellow dust, composed of spores, which may be rubbed off on the finger. Some insects cause similar, but dustless, spots.

Red spots are found in some rusts (æoidial stage of black rust of wheat), Chytridiaceæ, and leaf parasites of cereals and sugarcane. Mites and some sucking insects may also cause them. In the leaf form of red rust of tea and other plants, the spots are raised and often turn a sickly white when old.

FIG. 20. Pallid spots on *Brassica campestris* var. *Sarson* caused by *Peronospora parasitica*.

Brown spots are much the commonest effect of the action of leaf-spotting fungi, and are the usual result of the cells being killed. At a later stage, many turn pale or straw-coloured, especially in the centre. A few brown spots are caused by insects and some are due to unknown causes.

Black spots are almost always an indication of fungus attack. They are found in late stages of rusts and in many Ascomycetes (e.g., *Phyllachora*, Fig. 95).

Concentric bands of colour are found, in different shades of brown, in spots caused by *Alternaria Solani* and several other species, and in brown, red, and yellow in *Cercospora longipes* on sugarcane. They are generally caused by fungi.



**SHOT HOLE.**—This name is given to the perforations caused by certain leaf parasites of limited action. At first a brown spot appears, but the diseased tissues are soon cut off from the rest of the leaf and ultimately fall out, leaving a round hole in the leaf. They are common in fruit



FIG. 21. Brown spots on cabbage caused by *Alternaria Brasstiae*.



FIG. 22. Shot hole of peach leaf.

trees, such as peach and plum, and are found in tea and some other plants, but rarely in field crops. The fungi concerned are chiefly species of *Cercospora*, *Phyllosticta*, and *Clasterosporium*. Insects occasionally cause similar wounds.

**SCAB.**—This is a condition caused by cracking of the outer layers of fruits or tubers, the broken skin becoming dry, flaky, indurated, or sometimes corky. The scabs of apples and pears are caused by *Fusicladium* (*Venturia*), that of peaches by *Cladosporium* and *Clasterosporium*, and potato scab by several parasites (*Oospora*, *Spongospora*, *Spondyloladium*, etc.), and also probably by other non-parasitic agencies. Citrus scab, which is prevalent in India,

is usually caused by bacteria. Eelworms may cause scabbing of tubers.



FIG. 23. Scab of apples caused by *Venturia inaequalis*. (After Salmon).

**CANKER.**—Open wounds, often of a spreading nature and sometimes surrounded by a raised, tumour-like margin, are found on woody stems and even sometimes on annuals like pigeon-pea. They are caused usually by parasites that attack the bark and extend as far as the cambium. The tissues outside this layer are killed and slough off, leaving a wound exposing the wood. The raised margins are the result of efforts at repair by the production of callus, which may again be attacked and destroyed before healing has progressed far. In some cases (pink disease of rubber) canker may encircle the branch and cause a “ringing wound” which kills the part above. In the tea seedling disease (which is doubtfully parasitic) new roots may arise from the upper swollen edge of the wound (Fig. 190). Most canker-producing fungi are Ascomycetes (*Neotria*, *Dasyscypha*, *Aglaospora*), but some are rusts (*Peridermium*, *Gymnosporangium*) and Hymenomycetes (*Corticium*). Cankers are also caused by insects (woolly aphid of apples), frost, and bacteria.

**OTHER WOUNDS.**—The last three conditions mentioned result in wounds. Other wounds are caused by various fungi that attack leaves, as the leaf-shredding caused by *Sclerospora graminicola*, and the irregular holes and marginal corrosion of brown and rim blights of tea. Cavities in woody tissues are caused by some tree parasites, such as *Trametes Pini* and *Polyporus Shoreæ*.

**ROTTING.**—Rotting is often caused by the *Peronosporaceæ* (*Phytophthora*, *Pythium*), where the green parts are chiefly affected; by such fungi as *Rosellinia* and *Hymenochaete*, where the roots are concerned;



FIG. 24. Canker of tea stem,  
× 3.

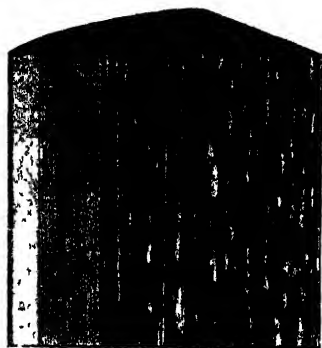


FIG. 25 Cavities in wood of *Pinus excelsa*  
caused by *Trametes Pini*, × 3.

by *Rhizoctonia*, where the region affected is usually the "collar"; and by various *Hymenomycetes* that cause wood rots in tree stems. Still more common are the rots of fruit and of various bulbs and tubers. The rot may be wet or dry, according sometimes to the parasite, sometimes to the host, and sometimes to various secondary or external conditions. Many wet rots are due to secondary saprophytic (putrefactive) organisms, chiefly bacteria, which follow in the track of the parasite (e.g., potatoes attacked by *Phytophthora infestans*). Rots of pulpy fruits (apple, tomato, and the like) are usually wet, owing to the quantity of cell sap present. They may be caused by weak parasites such as *Penicillium*, the vitality of the gorged cells being low (orange blue mould). Rots of woody and pithy stems are often dry, especially in the later stages (red rot of sugarcane, and various tree rots).

**FLUXES.**—Several tree diseases are characterised by an exudation from the bark of the stem. The nature of the exudation varies in

different cases. In the stem bleeding disease of the coconut in Ceylon, a colourless or brown, somewhat viscid liquid, rich in sugars and products of decay of the tissues, oozes out from cracks in the stem. In rubber canker, there is an exudation of latex in the older stages. Resin is poured out in conifers attacked by various fungi (*Peridermium*, *Fomes annosus*). Gum is found on the surface of the diseased parts in the foot-rot of citrus fruit trees. Exudations may also be caused by wounds and other non-parasitic causes.

**DROPPING OF LEAVES AND FRUIT.**—The leaves are shed as a result of the action of leaf parasites (*Cercospora personata*, *Hemileia vastatrix*)

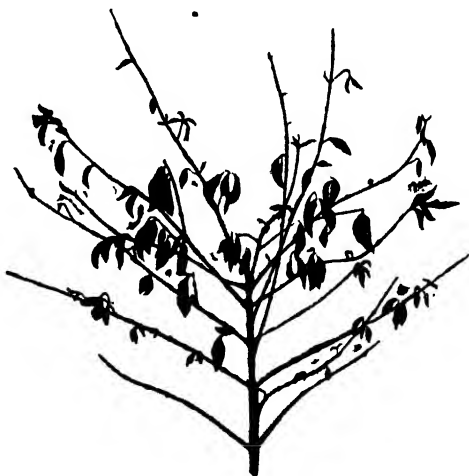


FIG. 23. Defoliation of coffee from twig disease.

and sometimes of stem or root diseases (coffee twig disease, pepper wilt). Dropping of fruit is caused by attacks of *Phytophthora* on palms and in some other cases. Leaves may also be shed after frost or in drought, and premature falling of fruit often causes much loss in orchards, apparently from some climatic influence not yet fully understood. The destructive dropping of cotton bolls seems also to be climatic.

**GALLS, TUMOURS, AND HYPERTROPHY.**—Abnormal outgrowths of the most varied character are often found on the herbaceous parts of plants



and also sometimes on woody stems and on roots and tubers. They may vary from tiny warts, involving only one or a few cells (*Synchytrium*), to



FIG. 27 Woody gall on *Acacia leucophylla* caused by *Haplophragmium ponderosum*,  $\times \frac{1}{2}$ .



FIG. 28 Hypertrophied and normal fruit of *Eurya acuminata* attacked by *Exobasidium Euryæ*,  $\times \frac{1}{2}$ .

the rounded tumours, several inches across, of maize smut (Fig 68) or the large woody outgrowths on *Acacia leucophylla*, caused by *Haplophragmium ponderosum*. The term "gall" is sometimes extended to include such conditions of abnormal development as are found in the ears of bajra or *Setaria* attacked by *Sclerospora* (Fig 81). Insects frequently form galls on plants, and a microscopic examination is usually necessary to determine the cause. In many cases, parasites cause a uniform increase in the size of an organ, not a one-sided or localised swelling such as those just mentioned. Such hypertrophy may affect whole organs, as in flowers attacked by *Cystopus candidus*, *Peronospora parasitica*, *Exobasidium Euryæ*, or *Eoasacus Pruni*; in the first of these often affecting all the floral organs, in the others only some of them (ovary, stamens). Or the hypertrophy may be confined to part of an organ (many branch hypertrophies, such those as caused by the *Eoasacaceæ*). A false appearance

of hypertrophy is sometimes caused by accumulations of fungus hyphæ or spores beneath the epidermis or bark, as in the huge, blister-like swellings found on *Casuarina* stems attacked by *Trichosporium vesiculosum*.



FIG. 29. Hypertrophied ovary of *Prunus Padus* attacked by *Exoascus Pruni*.

"WITCHES' BROOMS."—This curious name is given by country people in Europe to irregular tufts of crowded twigs, found in some trees and shrubs. The branch from which these arise is often swollen, and the shoots usually all turned upwards, short, and with small leaves. They are caused by parasitic fungi with perennial mycelium in the affected branch. The growth in length of the branch is often checked, and dormant buds are stimulated into throwing out new shoots each year, along which the fungus passes, and on which it may develop its spores. When spore production is finished, the leaves, and often the younger twigs, die off, to be replaced by a new crop next year. They are chiefly caused by *Exoascaceæ* and rusts, common examples being those on barberry bushes caused by *Æcidium montanum*, and on deodar caused by *Peridermium Cedri*, in the Himalaya. Mites and insects may cause similar, though more rosette-like, formations.

DEVELOPMENT OF DORMANT, RUDIMENTARY, OR NEW ORGANS.—Dormant buds are stimulated into growth by many other parasites besides those of the witches' brooms, though usually the action is indirect and



FIG. 30. Witches' broom on deodar caused by *Peridermium Cedri*. (After Troup).

caused by the death or weakening of branches higher up, as in mango trees attacked by *Cephaeleuros Mycoidea* on the twigs, or by defoliation, as in roses attacked by the common leaf parasite *Diplocarpon (Actinonema) Rosa*. New shoots are formed from the base of the plant in rice attacked by *Sclerotium Oryzae* (Fig. 88), and in the tea seedling disease, (which, however, may be non-parasitic). The rudimentary stamens in



FIG. 81. Sprouting of dormant buds of mango attacked by *Cephaeleuros Mycoidea*.

pistillate flowers of *Lychnis dioica* become fully developed when attacked by *Ustilago violacea* in Europe, except that the pollen is replaced by the spores of the fungus. Ovaries are developed in staminate flowers of *Buchloe dactyloides* infected with *Tilletia buchloeana* in America.

New (adventitious) formation of organs is found on leaves and stems, as a result of parasitic action, in several cases. One of the most remarkable

is the much-branched, leaf-like outgrowth from the leaves of a fern (*Pteris*) infected with *Taphrina Laurencia* in Ceylon. Leafy shoots have also been obtained by inoculating tobacco leaves with *Bacillus tumefaciens*, the crown-gall organism. The rust, *Cecoma deformans*, causes pallid, leafless, branched structures to appear on shoots

and leaves of a Japanese conifer (*Thujaopsis*). These are unlike any normal organ of the host. The galls formed by *Ustilago emodensis* on *Polygonum chinense* in Java and India seem also to be quite new organs. On the other hand, the supplementary petals produced in *Viola*

*silvestris* under the influence of *Puccinia Viola*, resemble the other petals and cause the appearance of double flowers. Tomatoes attacked by *Bacillus solanacearum* may produce intumescences on the stem, these seem to be arrested adventitious roots.



FIG. 32. Leafy outgrowth on frond of *Pteris* caused by *Taphrina Laurencia*. (After Glessenhagen).

**PROLIFICATION.**—By this is meant the continued development of a part after it has reached the stage at which it normally ceases to grow.



FIG. 33. 1, proliferation of spikelets of *Pennisetum* caused by *Sclerospora graminicola*; 2, leaf-like stamen from same,  $\times \frac{1}{4}$ .

It occurs in the ears of bajra and *Setaria* attacked by *Sclerospora graminicola*, the central axis of the flower growing on into a stunted leafy shoot, surrounded at the base by the glumes and stamens (Fig. 33, 1). In maize attacked by *Ustilago Zeae*, proliferation may take place in the interior of the carpels.

**TRANSFORMATION OF ORGANS.**—This is found chiefly in flowers and results in the change of one kind of floral leaf into another or into ordinary leaves. The stamens may become leafy in bajra affected by

*Sclerospora* (Fig. 33, 2). *Cystopus candidus* may cause petals to become like sepals, the stamens may have some of the characters of carpels, and the latter may be leaf-like; while in the flower of the Japanese plum, *Prunus Mume*, all the organs may be replaced by foliage-leaves, under the influence of *Cæoma Makinoi*. To this alteration of floral organs to leafy structures the term "phyllody" is applied.

ALTERATIONS IN SYMMETRY AND HABIT.—The common Indian weed, *Ipomæa reniformis*, bears radial shoots instead of the usual dorsi-ventral ones, when attacked by *Cystopus Ipomoeæ-panduranæ*. The short, unbranched stem with radical leaves of *Launea asplenifolia*, becomes an elongated, much branched axis with cauline leaves, when infected with *Puccinia Butleri*. Leaves may be changed from simple to irregularly lobed (*Berberis buxifolia* attacked by *Aecidium Jacobsthalii* in South



FIG. 24. *Launea asplenifolia*: normal plant below; plant attacked by *Puccinia Butleri* above,  $\times \frac{1}{2}$ .

America). Leaves and stems may be deformed, twisted, or otherwise distorted, under the influence of many parasites (e.g., *Cystopus candidus*). Single flowers may be altered from the regular (actinomorphic) to the irregular (zygomorphic) symmetry and vice versa (as in *Matricaria*

*inodora* attacked by *Peronospora Radii* in Europe, where ligulate flowers may be found in the centre of the disc and tubular flowers at the margin). The whole inflorescence may also be changed in type (e.g., the normal capitate inflorescence of *Acacia eburnea* becomes a spike when infected by the edible rust, *Æcidium esculentum*, found near Poona). Internodes may be lengthened (*Euphorbia cyparissias* attacked by *Uromyces Pisi* in Europe), or shortened (maize affected with downy mildew in Pusa). Branching may also be influenced, being reduced (*Uromyces* on *Euphorbia*), or increased ("witches' brooms"). In a few cases, changes occur which have been interpreted as reversions to earlier types in the ancestry of the host, as the formation of grain in the male inflorescence of maize attacked by *Ustilago Zeæ*, the spiral arrangement of the floral organs sometimes caused by *Cystopus candidus*, and the development of short axillary branches, ending in a small inflorescence, in broccoli infected with *Peronospora parasitica*.

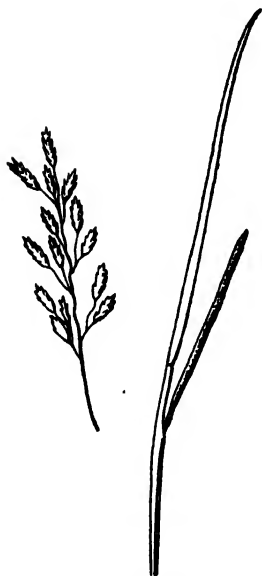


FIG. 35. *Eragrostis tenuifolia*: normal ear and ear atrophied by attack of *Epichloa cinerea*, nat. size.

**ATROPHY.**—Parts or organs are arrested in growth or even entirely suppressed, as a result of the action of many parasites. The same fungus may cause hypertrophy in one part and atrophy in another (*Peronospora parasitica*). The whole plant may remain stunted from early attacks of mildews, rusts, and other fungi. The leaves may be atrophied in "witches' brooms," and as a result of the actions of some rusts and species of *Synchytrium*; flowers, in poppy attacked by *Peronospora arborescens*, grasses with *Epichloë*, and some cereals with *Solerospora*; fruit, in rusted cereals, and from the smuts that infest the ovary. All these changes can also be caused by insect attack and several of them by unfavourable climatic conditions, poor soil, and the like.

Naturally, several of these symptoms may be found together. Thus in peach leaf-curl, the upper part of the twig may be swollen, the internodes shortened, and the affected leaves thickened and distorted.



FIG. 36. Peach leaf-curl.

#### EFFECTS OF PARASITIC FUNGI ON THE TISSUES OF THE HOST: MORBID ANATOMY.

There are two main ideas to be borne in mind, in considering the effect on the attacked plant of its parasite. On the one hand, we have not alone parasites capable of directly killing the invaded tissues and absorbing their contents, but others that have the power of stimulating the activities of the plant in the directions most satisfying to their needs: they can force the plant to manufacture new stores of food-material for their own uses, to form tissues rich in water, to develop thin-walled cells, easily penetrated by the parasitic hyphae, in place of normal thickened walls, and so on. On the other hand, the invaded plant develops active powers of defence. These may be direct, as when cork is formed to cut off the affected patch of leaf in infection by shot-hole fungi, or a cell wall thickens to resist the penetration of a hypha in beets attacked by *Sclerospora*; or they may be indirect, as when new leafy shoots are developed from dormant buds to replace those killed by the parasite in "witches' brooms." The net result, even in the latter case, may be entirely harmful, when, for instance, a turnip root stores up vast quantities of food at the expense of the other parts of the plant to meet the demands of *Plasmidiophora Brassicae*, or the hyper-activity of a "witches' broom" starves adjacent parts; or they may be in part successful, as in maize, which is able to supply all the needs of the maize smut by the production of a local hypertrophy rich in nutrient tissue, while manufacturing sufficient additional food to ripen a large proportion of its own grains successfully.



Besides those changes in the host plant which directly result from the conflict with its parasite, there are others which are due to the complex relationships which exist between the different functions of plants. It has already been remarked how alterations in food supplies can cause fungi to produce different kinds of spores. The same sometimes occurs as a result of the attacks of parasites on fungi, as in the *Mucor*, *Phobotus Kleinsii*, where infection by a Chytridiacean parasite causes the suppression of sporangia, and the production of zygospores which are normally rare. Something of a similar kind occurs in higher plants, where the suppression of flowers or seeds, or such conditions as the formation of an excessive number of petals or of petaloid stamens, may result from the influence of certain parasites.

The war between invading parasite and attacked plant is often subtle and long drawn out. It is natural that far-reaching effects should be produced on the structure and characters of the plant tissues and organs. All the modifications to be described below in the bodies of diseased plants may, however, probably be referred to one or other of three sets of factors: the direct influence of the parasite in modifying the attacked plant to suit its own needs; the defensive or blind reactions of the attacked body; and the secondary effects on reproductive and other parts, of the functional disturbance caused by the invader.

The changes caused in the tissues may be considered in their effects on single cells and on larger masses of tissue.

### EFFECTS ON SINGLE CELLS.

CONTENTS.—The cell may be killed without reacting against the attack. There may be no increase in size, nor change in the nature of the contents, except such as results from their death, and consumption by the parasite (Phytophthora). The effect may vary according as it is more vigorously exercised on one part of the cell contents than another. Sometimes the liquid of the cell is rapidly removed, and the normal appearances of wilting from drought (or "plasmolysis") are caused (several fruit-rotting parasites); sometimes chlorophyll is early reduced, leading to the appearance of pallid spots (*Aecidium Urticæ*, *Peronosporaceæ*); more usually, the whole cell contents collect towards the centre (Fig. 37, 1) in a grumous mass, which turns brown, apparently from the oxidation of substances like tannin present in the cell, and breaks up into amorphous granules often with the formation of refractive brown globules. These have been mistaken for parasites ("Pseudoomis") on several occasions. Starch may be directly attacked (*Polyporus ignarius*, *Phytophthora*) or may be left unaltered, especially where the cell has been rapidly dried out (*Guignardia Bidwellii*, *Venturia inæqualis*). Several of the potato-rotting species of *Fusarium* can hydrolyse gelatinous starch, but have no effect on the unbroken starch grains of the tuber.

Sometimes a reaction takes place within the cell. The nucleus may enlarge or become lobed or even fragmented (*Amphicarpæa* attacked by *Synchytrium decipiens* (Fig. 37, 2), *Capsella* with *Cystopus candidus*). The whole cell contents may increase, the membrane usually expanding at the same time (single cells affected with *Synchytrium pyriforme* or *S. puerariae*, superficial cells of grape leaves with black rot). Chlorophyll may be increased (*Synchytrium pyriforme* and *viride*) or formed in cells which usually contain none (*Cystopus candidus*). It may also persist longer than usual, and lead to the retention of the green colour of affected parts after the rest has withered (apple with *Venturia inæqualis* sometimes). This last fungus may cause an increase in the formation of crystals of calcium oxalate in the cells, while the acridal stage of *Puccinia coronata* may prevent their formation in *Rhamnus Frangula*. Starch may be greatly increased under the influence of *Cystopus candidus*, or may be found collected in tissues usually free from it. The formation of the red or purple pigment, anthocyan, within the cells, especially on the side towards the sun, is a frequent accompaniment of parasitic attack

(Synchytrium, Exoascus, Exobasidium) and often leads to coloured areas at or around the affected point. Most of the above reactions seem to be blind, or at least not of direct service to the plant in preserving it from injury. In a few cases, however, a directly

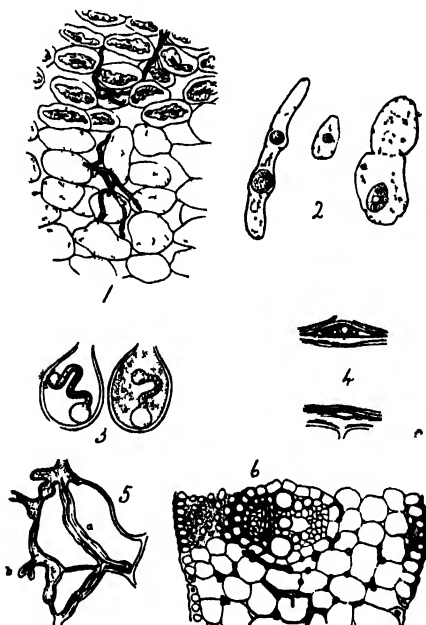


FIG. 37. 1, effect of *Phoma aptecola* on cells of celery bulb, those below are healthy. 2, alteration of nuclei of *Amphicarpae* by *Synchytrium decipiens*; normal nucleus in centre. 3, solution of cellulose wall of sporangium of *Phytophthora palmivora* by germ tube from a zoospore. 4, outer wall of rose leaf with hyphae of *Diplocarpon Rosa* below cuticle; below, the same showing the cavity dissolved in advance of the tip of the hypha. 5, hyphae of *Sclerospora graminicola* showing cellulose sheaths around intercellular hypha a and penetrating hypha b. 6, the same showing their failure to penetrate bundles or sclerenchyma. (1 after Klebahn, 2 after Kusano, 4 after Ducomet).

defensive effort is observable, the cell seeking to check the entry of the hypha by depositing cellulose around the threatened point, or by forming a cellulose sheath enclosing the hypha after entry [*Ustilago Zea*, *Sclerospora* (Fig 37, 5)]. In the cells of some fruits and green parts of plants, a tannin like body, toxic to fungi, is formed when a parasite gains an entrance. This arises from the action of an oxidase on substances such as gallic acid present in the cell, and so long as the oxidase is in sufficient quantity little injury results. As the parts ripen, the oxidase diminishes and the tissues lose their resistance

**MEMBRANE.**—Here again it is possible to distinguish between the directly destructive action of the parasite and the defensive or blind reaction of the host.

In destroying cell membranes, parasitic fungi make use principally of enzymes, of which they command a wide choice, differing, however, to some extent according to the fungus. The action has been most fully studied in certain parasites of woody tissue and in some leaf parasites which affect chiefly the epidermal and cuticular walls.

The most important constituent of plant membranes is cellulose, which forms the bulk of the walls of the softer tissues and of all young parts. Most parasitic endophytes are capable of dissolving cellulose by means of an enzyme (cystase or cellulase) (Fig. 37, 3). Sometimes the power is restricted to certain organs, or stages in the development of the parasite, as in the intercellular parasites that only penetrate cells by haustorial branches and in some Peronosporaceæ that traverse the epidermal cells by their germ-tubes, but are subsequently confined to the intercellular spaces (*Phytophthora Colocasiæ*). In other cases, the tips of all the hyphæ can dissolve a passage into the cells and extend readily from cell to cell (*Pythium de Baryanum*). In these, and in many other instances, the attack on the membrane is limited to a small area around the tip of the penetrating branch, sometimes only just enough to allow the latter to bore a narrow passage (Chytridiaceæ), sometimes larger, the membranes being swollen and softened for a little distance beyond the area actually in contact with the fungus (*Phytophthora palmivora*). It is evident too that enzyme production is limited to the tip of the hypha, since the hole in the wall does not subsequently enlarge. Many parasites of this type are unable to enter tissues with modified cell walls, such as the lignified cells of the vascular bundles (Fig. 37, 6), from which we must conclude that they do not possess the power of forming enzymes capable of dissolving such walls (*Pythium de Baryanum*, Sclerospore).

In the above cases, the action is confined to opening a passage into the cells in which the fungus feeds, and the nutrition obtained from the wall is trifling in amount. In another group of cases, the action is more extensive and the substance of the wall is largely consumed. In *Diplocarpon Rosæ*, a common leaf parasite of roses in India, and in several European forms (*Fusarium Hordearium*, *Venturia circinans*), the fungus grows at first entirely in the cellulose portion of the outer epidermal wall, which it corrodes to a greater or less extent, and no doubt consumes after transforming into sugars, though some of its food is probably taken by osmosis from the underlying epidermal cells since these are ultimately killed (Fig. 37, 4). Still more striking is the action of several parasites of the woody parts of trees. In some of these (*Polyporus Schweinitzii*, *Fomes carneus*, *Poria vaporaria*) the fungus extracts almost all the cellulose from the walls of the affected tissues, setting free the combined part (hadromal) of the woody impregnation. Ultimately, a brown, brittle mass resembling charcoal is found. The similar substance formed in the dry rot of timber, caused by *Merulius lacrymans*, has recently been shown to consist of humin bodies. As *Polyporus Schweinitzii* and *Fomes carneus* develop freely in the heart-wood, the cells of which are dead and contain mostly resin, pigment bodies, and tannin, it is evident that these fungi must obtain an important part of their food from the walls, and the disappearance of the cellulose indicates that this substance is largely made use of. Where wood gum (xylan) is present in the wall, it also may sometimes be utilised, as it has been found that certain fungi (*Glomerella cingulata*, potato-rotting species of *Fusarium*) possess pentosanases which can break down the pentosans (to which xylan belongs) into their component sugars. Wood gum is, however, highly resistant to other fungi (*Fomes nigricans*), so that pentosanases are not of constant occurrence.

Besides cellulose, the cell wall usually contains pectin bodies which are also carbohydrates (or allied bodies) and may be acted on by ferments (pectinase), being dissolved, with the formation of simple sugars. The primary cell wall consists largely of pectic substances, which persist as the middle lamella of the wall between two adjoining cells, and forms a sort of cementing substance holding the individual cells of the tissues together.

Pectin bodies are also found in the secondary parts of the cell wall lying next inside the middle lamella, but are scanty or wanting in the tertiary layer, which bounds the cavity of the cell and consists, at least when young, of pure cellulose.

The pectin bodies of the cell wall seem to be particularly liable to destruction by the action of parasitic fungi. Most intercellular endophytes force their way between the

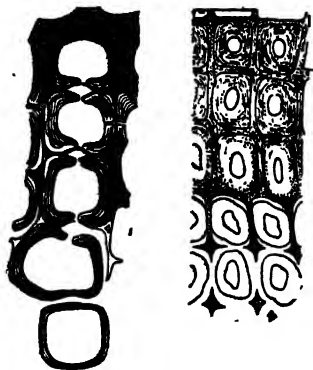


FIG. 38. Action of *Fomes annosus* (left) and *Trametes Pini* (right) on wood of pine and spruce. The lignified walls (dotted) are gradually delignified and the middle lamella dissolved, leaving the isolated cells (below) with walls of pure cellulose. (After Hartig (diagramatic) and v. Schrenk).

cells by dissolving the middle lamella, though in the majority of the parasites of parenchymatous tissues, such as the *Peronosporaceae* and rusts, the action is usually limited to the immediate neighbourhood of the hypha and the cells do not become separated from one another. On the other hand, in a few parasites of soft parts (*Botrytis cinerea*) and in several that attack woody tissues (*Trametes Pini*, *Fomes annosus*), the middle lamella may be entirely dissolved, so that the cells fall apart before the cellulose portion of the wall is destroyed (Fig. 38). Many saprophytic organisms also have this power of dissolving the binding substance of the cells, and they are made use of in the retting of jute and flax. It has been shown that certain moulds (*Rhizopus nigricans*) can utilise the pectic bodies, thus transformed into sugars, as food, and some of these organisms have little or no action on cellulose or the living contents of the cells.

Many cell walls are changed in composition as development proceeds, the changes affecting both the cellulose and

pectic portions of the wall. The most widespread of these secondary alterations is the process known as lignification, which appears to consist, in part, of a chemical combination of cellulose with an aromatic aldehyde "hadromal," and in part, of the deposition within the wall of an encrustation of complex bodies, from which the glucoside coniferin and the aromatic body vanillin have been obtained.\* All the cell walls of the wood (xylem) become lignified as they develop and the process also occurs to a greater or less degree in other tissues (sclerenchyma). The middle part of the wall (secondary layer) and the primary layer (middle lamella) are chiefly affected by these changes, the tertiary layer, bounding the cavity of the cell, often retaining its cellulose character to a considerable extent.

Lignified walls are acted on by many fungi. Some cause the decomposition of the hadromal-cellulose compound, using up the cellulose and setting free the hadromal, which may be extracted in quantity equal to that found in healthy wood and is therefore evidently not consumed by the fungus (*Fomes carneus*, *F. pinicola*). Others, such as

\* The nature of this "ligno-cellulose," to which the special properties of wood are due, is still problematical. All that Hesse and Hill ("Chemistry of Plant Products," 1913) feel justified in saying is that the cellulose is combined with at least two non-cellulose constituents, one of which appears to contain an aromatic nucleus and the other contains a furfural-yielding complex and is probably a pentosan. To both together, the name "lignin" is sometimes applied.

*Trametes Pini*, which is a destructive pine parasite in the Himalaya, cause a progressive delignification of the wall, beginning from the tertiary layer and working outwards to the middle lamella (Fig. 38). All the wood substances are removed (? consumed by the fungus) and approximately pure cellulose is left; later on, even this may be dissolved, and holes in the wood are formed. The enzymes which produce these actions are hadromase, which decomposes the hadromal-cellulose combination, setting free the hadromal (*Merulius lacrymans*), pectinase which dissolves the middle lamella, emulsin which decomposes coniferin, cytase which attack the cellulose, and doubtless others. They must often be produced in considerable quantities, since the action may be simultaneous over a fairly large area. It is probable also that their production is not limited to the tips of the hyphae, since holes may be found in the walls larger than the diameter of the penetrating hyphae (*Lentinus lepideus*). Different fungi utilise different enzymes, so that, for instance, *Bulgaria polymorpha* can remove the lignin bodies and dissolve the pectate of the middle lamella, but has no action on cellulose: *Fomes juniperinus* does the same, but ultimately destroys the cellulose also: *Fomes tisanus* corrodes the wall uniformly, from within out, without altering the relative proportion of lignin and cellulose, but does not attack the middle lamella: *Fomes nigricans* first dissolves the inner cellulose (tertiary) layer, then removes the hadromal and later the other lignin substances of the middle (secondary) layer, and, soon after, of the middle lamella; finally, it consumes the cellulose from within outwards, sometimes leaving the middle lamella (pectate) for a time, after the rest has been destroyed. Wood gum is not readily acted on by this fungus, and several others, and may remain in the cells and encrusting the walls when destruction is far advanced. All kinds of cells may not be equally acted on: thus the cells of the medullary rays may be early attacked and disappear long before the wood cells are entirely gone (*Polyporus subacidus*, *Fomes rimosus*); or the latter may be much altered while the medullary ray cells are still intact (*Polyporus sulphureus*); while the weakly parasitic *Polystictus pergamenus* leaves the vessels and medullary rays unaffected, after the wood fibres are rotted; and *Polyporus tisanus* confines its action to the fibres, so as to cause holes between the vessels and the parenchyma. These actions probably depend on differences in the composition of the cell walls or cell contents, since it has been shown that *Polyporus squamosus* rots the less lignified wood fibres of the sycamore before the more highly lignified vessels and medullary rays, while *P. tisanus* destroys the heavily lignified fibres and leaves the vessels and parenchyma alone, possibly because of their high tannin content.

The woody tissues are also often differently acted upon, according as they belong to the heart-wood or the sap-wood of the trunk, or, in the annual rings, according as they are the looser spring wood or the denser late summer wood. Many species are known that attack heart-wood only (e.g., *Stereum subpileatum*), or if they spread to the sap-wood, do so only in late stages, when the injury to the heart-wood has lowered the vitality of the living adjacent tissues. In these cases, the fungus usually arises from wounds, or insect burrows, involving the heart-wood, and the host is not liable to attack when young, before heart-wood is formed. Other species attack the sap-wood only, and their growth is arrested as soon as heart-wood is encountered (*Stereum hirsutum*, *Polyporus Shorea*) (Fig. 39). Some, such as *Polyporus subacidus*, destroy heart-wood and sap-wood with equal rapidity, while *Polystictus versicolor*, on the liliae, begins in the heart-wood, often from borer holes, and when a footing is obtained, grows outward towards the bark, through the living sap-wood. It has been shown that in many of these cases, the amount of air present in the tissues is the determining factor, and that this is, in turn, dependent on the quantity of water in the wood and the relative proportion of solid matter, such as cell walls, to the cell cavities. Many wood-destroyers have a large air requirement, others seem to require more water. *Stereum hirsutum* is believed to be unable to flourish on pine heart-wood, because the closing of the tracheids and excessive resin prevent aeration, while *Polystictus versicolor* penetrates best into liliae sap-wood, when this has been dried out by borers. Again, *Polyporus subacidus* attacks the summer wood of the spruce

sometimes, but leaves the spring wood alone (Fig 40), whereas *Lenzites sepiaria* (a timber saprophyte) rots the spring wood, without (in the early stages) injuring the more



FIG. 39. Effect of *Polyporus Shoreae* on sap wood of *Shorea robusta*, nat size.

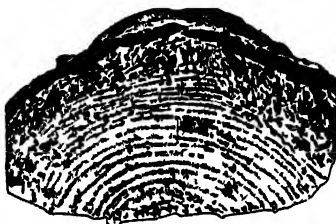


FIG. 40. Effect of *Polyporus subacidus* in delignifying the summer wood of each annual ring of spruce. (After v. Schrenk).

compact late summer wood Tests with this last fungus seem to indicate that it has enzymes capable of destroying hadermal and coniferin but not vanillin In several cases (*Trametes Pini*, *Polyporus Shoreae*, *Stereum frustulosum*), the destructive action is visible in "pockets" of varying size, the intermediate spaces being but slightly altered, though all are equally within reach of the hyphae.

In the higher plants, the exposed surface is usually clothed by a continuous non-cellular membrane, the cuticle, which lies over the epidermal cells. This layer is formed by the epidermis at an early age, but opinions differ as to whether it is derived from cellulose or pectic bodies by the subsequent deposition of cutin, like the lignin of lignified walls, or is a non-cellulose independent layer. Cutin is not confined to the cuticle, but is often found impregnating the deeper layers of the outer epidermal wall and even in the lower walls of the epidermis and in the sub-epidermal cell walls, to form what are known as outinised walls. After removal of the outin in these cases, cellulose and pectin substances are left. Cutin is considered to be of a fatty nature, and is almost impermeable to water and gases.

Several fungi are capable of dissolving cutin and of decutinising cutinised membranes.

In those parasites that penetrate directly into the epidermal cells from the surface, a passage is bored through both the cuticle and the outer wall of the epidermal cell; in those that live in or below the cuticle, only this layer is pierced, and the mycelium continues its development in the thickness of the wall (*Venturia inæqualis* (Fig. 41, 1)). The sub-cuticular parasites are sometimes capable of considerable growth horizontally, and may form extensive plates of mycelium within the outer walls of leaves (*Exoascoa*, *Diplocarpon Rosæ*, *Venturia*). Some seem to progress chiefly by forcing their way mechanically between the layers of the wall (*Stigmatea Robertiani*, *Guignardia Bidwellii*), others by digestion of the membrane (*Cyclogonium oleaginum*, *Fusicladium Pruni*). In the latter group, some attack only the outinised parts (*Cyclogonium*), others both outinised and cellulose parts (*Fusicladium* sometimes), others the cellulose parts only (*Venturia circinans*). When outinised membranes are attacked, the first stage is usually a decutinisation, just as in several parasites of woody tissues the first stage is a delignification. The action on the membrane is usually localised to the parts around the filament to a greater degree than in the parasites of woody tissues previously described (Fig. 41, 2).

Another type of wall is found in many plants, especially in the familiar cork of the bark of trees. This is the suberised cell wall, in which the essential substance is a fatty body "suberin," which is allied to cutin and, like it, impermeable to water and gases. In cork cells, the suberin is deposited in a layer bounding the middle lamella, the latter becoming lignified at the same time. Inside the suberin layer, a layer of cellulose may

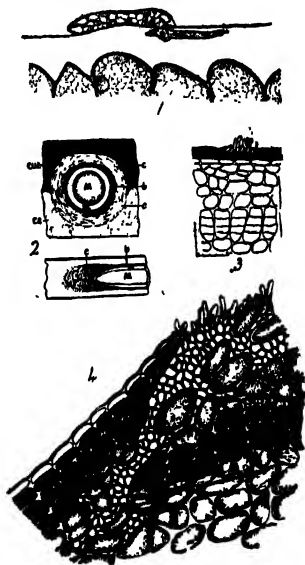


FIG. 41. 1, infection hypha of *Venturia inæqualis* entering cuticle of apple fruit; 2, diagram of digestive action of a hypha on the outer membranes, in transverse and longitudinal section: *m* hyphal cavity, *m* hyphal wall, *d* brown products of digestion, *c* zone of digestion, *cut* cutinised part, and *cc* cellulose part of membrane; 3, formation of reactionary cork in apple twig attacked by *Venturia inæqualis*; 4, penetration of the reactionary cork formed in pear twig attacked by *Venturia pirina*. (1 after Wiltshire, 2-4 after Ducomet).

finally be deposited, bounding the cavity of the cell, but cellulose seems to be absent from the suberin layer itself. Cork cells are usually formed as a distinct new tissue by the division of embryonic cells, but cases also occur not infrequently of the suberisation of pre-existing cells, in a manner similar to the lignification of woody cells, except that, while lignification begins as a rule in the middle lamella and adjoining parts of the secondary layer of the wall, suberisation begins on the inside of the wall, bounding the cell cavity. Both processes may occur in the same cell, lignification usually preceding suberisation.

Owing to the impermeability of suberin, the tissues external to the suberised layer die and are often gradually shed. This occurs normally in the bark of trees. Also if, for any reason, the plant requires to reduce loss of water, such as occurs when the cuticle is destroyed, or a wound removes the impervious outer bark of a twig, cork is formed below the injured area. This reactionary cork is frequently developed as a result of damage done to the outer layers by parasitic fungi, and is one of the most valuable defensive reactions against parasitic attack (Fig. 41, 3). Not only is the continued extension, in depth, of the parasite checked (suberin being particularly resistant to attack by fungus hyphae), but the external tissues, and with them often the parasite itself, are killed and shed. Shot hole (see p. 81) is one of the best examples of successful defence in this manner, each hole being due to the formation of a ring of cork around the infected leaf area. Unfortunately the plant is often unable to form a continuous corky barrier in time

to prevent penetration at every point, and the mycelium gets below it and continues to kill cells deeper in (Fig. 41, 4). A new layer of cork may then be formed, and the process may be repeated several times. The result is the formation of an irregular wound with scaly margins, such as is found in the well-known scab of apples and pears and in certain twig diseases, such as red rust on tea and mango. All living cell-tissues are capable of reacting in this way—leaves, tubers, fruits, and the outer tissues of stems especially. Whether the reaction is effective or not, depends in general on the vigour of the host and the rate of growth of the parasite. The process is most commonly found in attacks by hemi-saprophytes, for, as already mentioned, many of the most highly developed parasites (*Peronosporaceae*, *rusts*, *smuts*) are so able to regulate their demands on the host as not to provoke any alteration that might impede their progress.

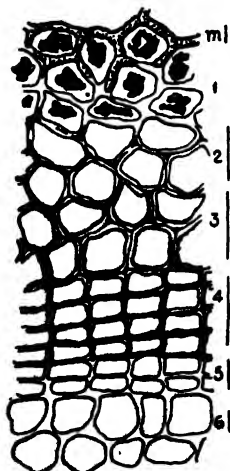


FIG. 42. Scheme of defensive reaction of cells: m, mycelium; 1, cells dead without reaction; 2, angular lignification; 3, ligno-suberisation; 4, cork; 5, meristem; 6, normal (After Buisson).

Other reactionary processes besides suberisation are often found. In leaves attacked by sub-cuticular parasites, the cellulose part of the outer epidermal wall may become cutinised below the hyphae (*Diplocarpon Rosa*) or lignified (*Cyclospium oleaginum*). When the hyphae run within the cutinised part of the wall, as the latter parasite does, the part internal to the hyphae may be lignified as well as cutinised. In the deeper walls of the epidermis, and in those of the tissues further in, the first reaction to a superficial or penetrating

parasite is often lignification. This may be followed by suberisation or the formation of a layer of cork cells at a greater depth. The parasite may also provoke the division



of the cells, which may enlarge somewhat first; division is by the formation of tangential walls (*Venturia inaequalis* on apple leaves, *Mycodermis cuticularis*), and is usually associated with suberisation. The intercellular spaces which are found in the deeper part of the leaf may be much reduced in size as a result of enlargement of the cells (*Venturia inaequalis*). All these changes seem to have as object the reduction of the loss of water by transpiration, which may be carried so far as to lead to the death of the outer layers and hence is not necessarily always beneficial.

Certain other effects of parasitic attack in which the membrane may play a part remain to be referred to. A gummy degeneration of the wall is found in several cases. Thus the walls of the stomatal cells of the galls caused by *Ustilago Zeae* on maize may

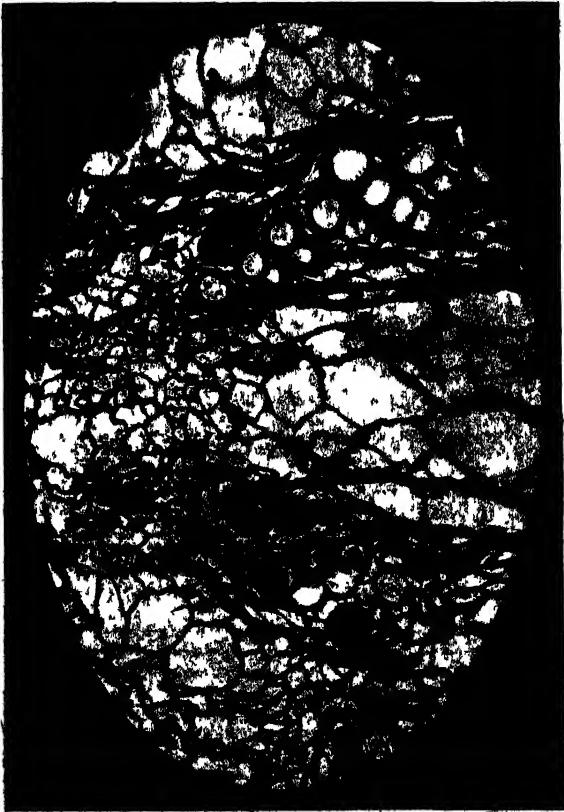


FIG. 43. Gum-formation in vessels of tobacco induced by *Basilline solanacearum*.

be partially changed to wound gum, and the same substance is frequently found in the water-carrying vessels of plants in the neighbourhood of wounds. A gum is produced in quantity in the vessels of tobacco plants attacked by *Bacillus solanacearum* and is probably derived from the walls. The gummosis of the peach, cherry, almond, citrus, and other fruit trees, which may be caused by fungi as well as by injuries and other agencies, is due to gummy degeneration of the secondary layer and middle lamella of the walls of the young wood cells in the neighbourhood of the cambium, and sometimes of other tissues. The gum may be produced in such quantities as to force its way to the surface, where it forms pearly drops. A mucilaginous degeneration of the middle lamella (pectic degeneration) often results from the attack of *Venturia circinans* on Geranium. *Exoascus Cerasi* also causes swelling and mucilaginous degeneration of the walls of affected tissues.

An increased production of resin in the wood of conifers is caused by the attack of certain fungi (*Peridermium Pini*, *Agaricus melleus*). The resinous contents of the walls are liquefied and flow up and down the vessels and outwards into the living parts, under considerable pressure, so that a resin flux is produced from the surface of the stem.

Another condition which may result from the attack of parasitic fungi is the formation of "thyloses," thin-walled cells found in the vessels of the wood as a result of growth of living cells of the medullary rays and wood parenchyma through the pits in the vessel walls. These protrusions are found normally in the neighbourhood of wounds, and serve to check the loss of water and prevent air entering. They are also produced in large numbers in areca palms attacked by *Fomes lucidus*, following infection of elm and other plants by *Nedria cinnabarina*, in Hovea with pink disease, and probably in many other cases.

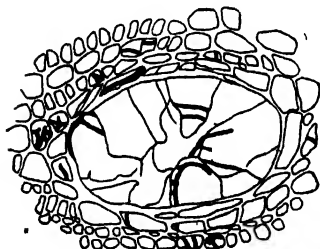


FIG. 44. Thyloses in vessel of Hovea affected with pink disease (*Corticium salmonicolor*),  $\times 80$ . (After Brooks and Sharples).

Finally, some parasitic fungi are capable of preventing the secondary modifications of cell walls in the neighbourhood of the infected area. Thus *Leptospheria herpotrichoides* causes the cells of the sclerenchyma to remain thin-walled, so that the culm is unable to stand erect, when it attacks wheat; *Cytospora candidus* prevents lignification of the medullary rays of Capeella, and *Ustilago Zea* of the vessels of maize; cutinisation of the outer wall of the epidermis does not occur in the tumours formed by the last-named fungus, and so on. Many of these changes are rather to be considered as tissue alterations, than as affecting single cells, and so belong to the next section.

Naturally, in many of the cases quoted above, the action is not confined to the cell wall, and the destructive potentialities of fungi, such as *Polyporus squamosus*, owing to the number of enzymes they can form, have already been mentioned (p. 53). In *Botrytis cinerea*, the wall is early attacked and the tissue cells are still alive after they have lost their cohesion and can even be separated from one another by gentle shaking (hair of *Tradescantia*). This is due to the enzymic action being first exercised on the middle lamella. Later, the rest of the wall is disintegrated. Death of the cells takes place after disorganisation of the walls has become strongly marked. It is said that the fungus is only able to continue its growth in the dead cells, and is therefore a saprophyte, in so far that it can only grow on dead substances, though capable of killing the cells in advance of its growth.

## EFFECTS ON TISSUES.

Parasitic fungi not infrequently act on more or less considerable masses of tissue, causing them, in some cases, to enlarge (hypertrophy), with or without a change in the characters of their cells, in others preventing their formation, in others again leading to the production of new tissues. Only a few characteristic cases need be referred to.

Very many instances of hypertrophy are known, involving almost every tissue and every organ of plants. As a rule, the cells of hypertrophied tissues show some differences in their character or contents from those of normal tissues. Hypertrophied roots are known in mycorrhizas and in "finger and toe" and some other root diseases. Hypertrophied stems occur in many plants attacked by rusts, Exosporium, and canker-producing fungi. Leaves are hypertrophied by rusts, Exosporium, Exobasidium, and many other fungi. Flowers are enlarged as a result of the attacks of some Peronosporaceæ (*Cystopus candidus*, *Sclerospora*), smuts, Exosporium, and Exobasidium.

The Myxomycete, *Plasmodiophora Brassicae*, which causes "finger and toe" in turnips and allied plants (a disease not yet recorded in India), enters probably through the root hairs and reaches the outer root cells. These are not killed outright, but are stimulated first, as is indicated by their growth in size, and division. The cambium shows extraordinary activity, as a result of which new, thin-walled cells are formed peripherally, in the position of the phloem, and in these transitory starch is accumulated. The vessels alone show lignification, but remain imperfect and pursue a tortuous course. The attacked root is soon converted into a large, distorted swelling, which finally breaks down and rots as the cell contents are consumed. The hypertrophy in this case is due to increase in the number and size of the cell layers external to the cambium.

Hypertrophy of the stem is well marked in the "witches' brooms" produced by species of Exosporium. The increase is chiefly in the bark, especially the hypoderm, where the cells are more numerous and larger, while their normal arrangement in longitudinal rows is lost. The cork cells are also enlarged and retain their protoplasm longer than usual. In the phloem, the fibres are small and with thin walls, while the sclerenchymatous cells are numerous, large, and also thin-walled. The medullary rays are enlarged and increased in number, but the rest of the wood is less affected, though its fibres have wide, often chambered cavities and slender walls, and the tracheae are more numerous. The pith also is larger than in the normal stem. In general, stem swellings are chiefly due to an abnormal increase in parenchymatous tissues, which may be formed directly from the cambium or by the growth or division of pre-existing tissue cells. This is often accompanied by a diminution in the walls of normally thickened elements such as sclerenchyma, and an increase in the cell contents, especially starch



FIG. 45. Section of stem of Jasmine attacked by *Uromyces Holstoni* (Air bubbles in the pith have been given unnecessary prominence by the Indian artist),  $\times 15$ .

(*Cystopus candidus*). The swelling is sometimes practically confined to the tissues external to the cambium (Jasmine with *Uromyces Hobsoni*).

In hypertrophied leaves, the extension of the influence beyond the limits of the cells actually invaded by the parasite is often marked. In certain Chytridiaceae, for instance, the parasite is only found in a single cell of the epidermis, but the cells in the neighbourhood are stimulated, divide, and form a little, wart-like outgrowth on the surface of the leaf. In the same way, some sub-outicular parasites (*Guignardia Bidwellii*, *Venturia inaequalis*) cause increase in the size of the leaf cells further in, and sometimes their division. In these cases, the normal differences between the upper and lower parts of the mesophyll (palisade and spongy parenchyma) are retained, the hypertrophy affecting the palisade cells chiefly. In other cases (*Cystopus candidus*), the whole of the mesophyll is converted into a tissue of large, thin-walled cells, with small intercellular spaces and numerous chlorophyll corpuscles, and the differentiation into palisade and spongy parenchyma is lost.

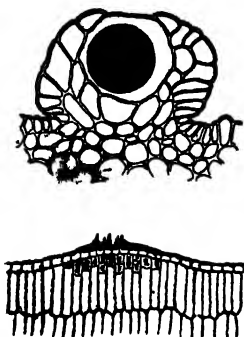


FIG. 46. Above: gall caused by *Synchytrium Mercurialis* on *Mercurialis perennis*,  $\times 100$ . (After Woronin). Below: hypertrophy and division of outer palisade cells in apple leaf attacked by *Venturia inaequalis*. (After Ducomet).

Flowers may be enlarged by the transformation of their parts (petals, stamens, etc.) into leafy organs (phyllody), or by increase in size without this transformation. Thus *Sclerospora graminicola* causes the stamens to resemble small leaves in shape and structure, while *Cystopus candidus* may cause an immense enlargement of the sepals, due to the development of a mass of thin-walled, starch-forming parenchyma, with a corresponding increase in the vascular and parenchymatous parts of the bundles, but not of the fibrous part.

Tissues that occur normally in healthy parts are sometimes prevented from developing owing to parasitic attack. Thus the thin-walled parenchyma of stem hypertrophies may replace the normal collenchyma, in plants attacked by *Protomyces macrosporus*. Sclerenchyma is similarly replaced by thin-walled cells in many cases. Cork formation is suppressed in twigs of hawthorn infected with *Ræstelia*. The absciss layer is prevented from forming in leaves of cherry attacked by *Gnomonia erythrostoma*, with the result that they do not fall in autumn, but remain hanging on the tree and serve to infect the new leaves in the following spring.

In a few cases, tissues are formed which do not resemble any normal tissue of the host. Thus the galls formed by *Ustilago emodensis* on *Polygonum chinense* contain, in the part which bears the spores of the fungus, a tissue of long, filamentous cells, in the meshes of which the spores are entangled.

## CHAPTER V.

### THE PRINCIPLES OF THE CONTROL OF PLANT DISEASE.

IN attempting to check diseases of plants, knowledge is usually required of the cause of the disease, of the life-history of the parasite, and of the circumstances which influence the establishment of ~~parasitic~~ relations between it and the host. Knowledge of the cause is of advantage, largely because it often allows of previous experience with the same or closely allied diseases being applied to specific cases ; treatment may thus be possible as soon as a diagnosis is made. Knowledge of the life-history of the parasite is clearly essential before all the methods of checking it can be tried ; such knowledge is available in many cases, once the cause is determined, but in India there is still much to be done before this can be said to be generally true. Knowledge of the circumstances which increase or diminish the power to injure of the parasite, and the resistance to attack of the host, is still elementary ; and these circumstances are so dependent on small differences in climate and other external conditions, on local agricultural practices and the like, that it is seldom possible to utilize rigidly the experience of workers in other localities.

Considered broadly, it is impossible to assign a greater degree of importance to any one of these requisites than to the others. The cause of malaria in man was known for a considerable time before the life-history of the parasite had been followed out ; and the value of the use of quinine in the disease, as evinced by its destruction of the parasite in the blood, has not been lessened by the subsequent discovery of the origin of the latter from mosquito bites, though this added to the methods of fighting it. The causes which lead to alterations in the severity of an attack are still obscure, but are not likely, when known, to reduce the significance of the earlier discoveries. Each step is dependent on what went before and none can be said to be the most important. Viewed in this light, the perennial controversy between those who believe that the parasite is the important factor and those who consider that attention should be concentrated on the host, loses much of its meaning. A knowledge of each is equally important when the study of the conditions influencing the severity of a disease is taken up, and its control is attempted.

In previous pages, it has been explained how fungi cause disease, and in the second part of this book the cause of the disease and life-history of the parasite will be considered in detail in a number of specific cases. It is proposed in the following section to deal briefly with the circumstances which influence the severity of a disease, or, in other words, which affect the establishment of the parasitic relation of the fungus to its host, a matter on which, as already said, there is far too little information available.

### **Conditions influencing the severity of disease. Virulence :**

#### **Predisposition : Immunity.**

THERE are two quite distinct sets of factors which control the intensity of any given disease. There are the circumstances which aid or check the parasite in its growth, multiplication, and spread : and there are those that make the host more or less capable of resisting the attack. Some of these are innate and not readily modified by the surroundings, others depend on external conditions and vary from place to place, from year to year, and with different agricultural practices.

CONDITIONS AFFECTING THE PARASITE.—The conditions which affect the parasite have already been partly considered when discussing dissemination and infection. Many are dependent on the external environment, sanitation, weather, type of cultivation, and the like ; others appear to be due to innate variations of virulence in the parasite. Of the first group, some which may be said to be influenced by general principles of plant sanitation may be given priority, as being subject to a certain measure of control.

The growth of large areas under a single crop assists in the spread of disease. In mixed vegetation, an infected plant is often not a serious danger to its neighbours, as the majority of parasites can feed on only one or a few species of host plant. On the other hand, a disease centre in a field bearing one crop may lead rapidly to infection of the whole field. Artificial inoculations with the potato blight fungus, in a locality in the United States where no disease was present naturally, have proved that two infected plants near together can spread infection sufficiently to destroy the above-ground parts in a half-acre plot in 29 days. This would only be possible where there was nothing to prevent the spores rapidly and surely reaching a suitable host plant. Hence the mixing of crops so common in India has its advantages from this point of view, for a field of, say, wheat, barley, gram, linseed, and peas mingled together,

will probably suffer less from parasites than five separate plots of these plants. A smaller percentage of the spores of the rusts of each crop will reach a plant that they can infect, in the former case, the gram wilt fungus will have further to travel in the soil before it meets another gram plant, and so on.

Rotation is a most useful means of checking the development of such parasites as can live or preserve their spores for a time in the soil. The wilt diseases so common in India, and those due to various species of *Rhizoctonia*, are only kept within bounds by this practice. A five years' rotation has been recommended in Europe for "finger and toe" of turnips, and eight or nine years' for flax wilt, while cabbage yellows cannot be controlled by any reasonable rotation. These are perhaps exceptional cases, three years being generally sufficient; but it is already known that the spores of the cotton wilt can live for more than three years in India in a dry condition, and since the aim is to keep the soil free from a suitable host until all the spores are dead, it is evidently unwise to grow susceptible cotton frequently in soil where wilt is prevalent.

Accumulations of dead leaves or debris from the crop are sometimes a sanitary danger. The *Ascomycetes* and *Phycomycetes* often ripen their resting spores under such conditions, and other fungi, such as the red and black rots of sugarcane, can continue to develop as saprophytes in dead stems and leaves, particularly of their host plants. Gram, arhar, and cotton stalks should similarly be destroyed when the crop is affected by wilt. In tea, coffee, and rubber estates, the removal and destruction of prunings and of the stumps and debris of jungle and shade trees when cut down, is now generally recognised to be a desirable sanitary precaution and must prevent the continued growth and reproduction of certain dangerous parasites. In a few cases, weeds harbour parasites that can pass to cultivated plants, but this risk is, as a rule, slight.

Since many parasites only produce their spores after the damage caused by them is well marked, there is no more effective method of hindering their dissemination than by the removal and destruction of the infected individuals or of their diseased members. To give a reasonable promise of success, two conditions should be fulfilled. The disease must be easily recognised, and it must be of such a nature as to admit of being readily and completely excised before the parasite has been widely disseminated; there is obviously little to be gained by removing a diseased part after it has liberated quantities of spores. The *Sclerospora*

disease of bajra may be taken as an example that meets these requirements, for though all attempts at reproducing this class of disease artificially have failed, there seems little doubt that it is carried over by the resting spores. It is easily seen, and recognisable at an early stage, and if all infected plants were pulled out and destroyed, little damage need be feared in following years. There is probably more room for this method of checking disease in India than in most other countries, as the generally small plots met with allow of more laborious methods than would be feasible where large areas are the rule. In some cases, as in the removal of smutted sugarcane, extensive co-operation may not be necessary, as the fungus does not seem to be capable of wide dissemination, is confined to certain varieties, and is often at a distance from other fields of susceptible cane. In others, as in the bud rot of palms, the efficacy of the treatment depends on no diseased trees being left, and the work in a village may be rendered useless in the long run if all do not unite. The cutting out of diseased parts is particularly applicable to fruit and forest trees, and such woody plants as tea, coffee, and rubber, as it is often possible to prune off infected members without causing much harm to the plant. Thus it is a regular practice on many estates to cut out pink disease of rubber, and canker and thread blight of tea. Mulberries used for silk-worm culture are much damaged in Kashmir (as in Europe) by *Polyporus hispidus*, but the loss can be reduced by cutting out the young fruiting bodies with the underlying diseased parts. The foot rot of oranges and some forms of canker in fruit trees may also be dealt with in the same way with success. In intensive forestry, the removal of the sporophores of certain tree parasites is a regular part of the operations in some countries, but has not yet been adopted in India; infected trees are not saved, but the dissemination of the disease is checked.

Many wound parasites are dependent on access to broken tissues of the host plant, especially those left exposed in woody plants by pruning or the breaking off of branches. The protection of these wounds in fruit and forest trees and woody crops is therefore of importance. In temperate countries, wood tar (Stockholm tar) is usually employed, but in the tropics it tends to become too liquid in hot weather and runs over the sound bark, which it kills. Coal tar is better, as it is more permanent, antiseptic, and not so liable to run. It should be applied with a small brush to the raw surface only, and not allowed to get on sound bark, which it will burn. In the United States, a thick coating with white-lead paint is recommended.



The use of cuttings and seed from diseased plants is sometimes very dangerous. This particularly applies to plants ordinarily propagated vegetatively by setts, cuttings, tubers, and the like. In many of these, the parasites ordinarily extend into the parts used for propagation and retain their vitality long enough to infect the new shoots (potato blight, red rot of sugarcane). In some diseases, the seed becomes contaminated and seed from diseased plants should be carefully avoided (anthracnose of cotton and bean, gram blight).

In a few cases, the use of fresh farm-yard manure is attended with risk, as the spores of such fungi as the cereal smuts may survive passage through the alimentary canal of farm animals fed on a diseased crop. In India, the danger from this source is slight, but old, well-rotted manure is preferable for most purposes.

Protective wind belts or "wind breaks," composed of trees or shrubs, may prove valuable in checking the spread of wind-borne diseases in certain crops, such as tea and coffee. It has been proved that artificial screens do exercise a definite effect in this direction. Experiments in tea gardens show that the bushes to leeward of the wind barrier suffer less from leaf diseases than those freely exposed to the wind. The method is worthy of more extensive trial in certain cases, but one danger in its adoption should not be lost sight of. Some parasites of tea and coffee, both insect and fungal, can live on plants that may be used as wind breaks. Thus it is certain that a number of the shade trees used in Assam tea gardens are liable to red rust, and it is not improbable that the parasite reappears on heavily pruned tea, from which it has mostly been cut out, as a result of reinfection from these trees or from the jungle.

In the storing of fruit, potatoes, and the like, considerable care is often required to prevent the maintenance of conditions favourable for the development of parasites that cause rotting. Most of these are weak parasites and require specially suitable surroundings to enable them to cause much damage. Excessive moisture and a high temperature are the chief of these, and act probably both by giving the parasite optimum conditions for its growth and spread, and by rendering the stored produce more susceptible to attack. Rough handling, of such a nature as to injure the protective surface layers, also aids weak parasites in the penetration, and it is well known that hand-picked fruit is less liable to rot than wind-falls, owing to the bruised areas in the latter case being very susceptible to infection. Actual contact in store also naturally assists the spread of decay, and for long-distance transit it is often advisable

to wrap each individual separately. The locality selected for storing should be dry, cool, and well ventilated. Fruit should be placed in a single layer, not touching. Potatoes are best stored in thoroughly dry sand, which, in places exposed to the potato moth, should cover them completely. None but sound tubers should be kept, as not only do rotting tubers increase the numbers of the rot-producing organisms, but they supply, while decaying, a considerable amount of moisture. The store should be picked over regularly to remove infected tubers.

Amongst the environmental factors that influence the development of parasitic fungi, moisture, temperature, light, and air are the chief. The density of a crop may affect all these, and this again may depend on the amount of soil moisture and the good or bad nutrition of the host. Since, as a rule, these factors act by influencing the germination of the spores, they have been already referred to in the section on infection. A few special cases may, however, be mentioned here.

Soil moisture and temperature are known to have a marked effect on the prevalence of some of the cereal smuts. In Europe and the eastern parts of North America, infection with bunt spores from the soil is uncommon, as the spores germinate in winter in wet soil when the temperature rises above 41°F., and if frost or drought then sets in, are killed. In the cold, dry areas of the north-west of America, however, the spores remain alive without germinating until the following sowing time, when they germinate with the wheat and may cause heavy loss. In flag smut in Australia, the spores similarly remain alive, without germinating, in dry seasons. If rain comes before the sowing time, they germinate and die out in the absence of a suitable host; but when the first rain is at sowing time, host and parasite germinate together and severe infection may follow.

In wheat rust, the determining factor in many parts of India is the humidity of the air in the early months of the year. Certain observations indicate that it is the humidity of the air within the crop that is of importance, and that this may vary considerably with the density of the crop. In some years, the parasite is much more prevalent where the crop is heavier; and there may be, in the same field, 20 per cent more moisture in the air within the crop in such parts than in those with a light crop where rust is not severe. The density of the crop in these cases is often a measure of the relative amount of soil moisture during the dry period of the year. Here, therefore, the ultimate factor may be the soil moisture though the immediate cause is the humidity of the air, which favours free

germination, and infection by the rust spores. Of course in very moist weather even light crops may be severely gusted; and the injury caused by a widespread rust epidemic may actually be greatest on light soil that dries out easily; but this is due not so much to a multiplication of the parasite as to lesser vitality of the host. There is also some evidence that, once infection has occurred, subsequent development within the plant is favoured by a low water content. Many other cases are known where atmospheric moisture is the limiting factor in parasitic attacks, but this must be distinguished from total precipitation, as heavy downpours of rain sometimes act beneficially by washing the spores from the plant into the soil.

The influence of light is perhaps best illustrated by the powdery mildews (*Erysiphaceae*). It is well known that many of these parasites develop most vigorously in shady situations. Wheat mildew in India has only been seen causing serious damage in half-shaded pot culture experiments, and tobacco mildew is rarely destructive except where direct sunlight is cut off. Vine, strawberry, and cucumber mildews have been noticed in other countries to behave in the same way, while in England, experiments with vegetable marrows showed that plants exposed only at night were soon covered with mildew (*Erysiphe Cichoracearum*) while those unprotected only during the day remained practically free, though a far greater number of spores is liberated by day than by night. The exact way in which shade favours these parasites does not seem to have been worked out, but it presumably promotes germination of the spores and infection of the host. On the other hand, shade has been reported to check celery leaf-spot (*Cercospora Apii*), asparagus rust, and the leaf disease of strawberries (*Mycosphaerella Fragariae*) in the United States. Shade also reduces chilli die-back at Pusa, because of the lesser deposit of dew in shady places.

Air is known to affect the growth of parasites in some cases. It has been established that some of the parasites of the wood of trees (e.g., several species of *Fomes*, *Stereum*, *Nectria*, and *Valsa*) are only able to grow when they are well supplied with air. The quantity of air present in the tissues depends on the water content of the wood, being highest when the water is lowest. It is also affected to a certain extent by the density of the tissues, that is to say by the relative proportion of cell walls to cell cavities. Hence these parasites attack most readily at periods when the wood is relatively dry (as during the winter rest of deciduous trees), and often spread more quickly in the loose tissue of the early-formed

portion of the annual rings than in the denser late-formed wood. It is possible that the reason why the wilt diseases caused by species of *Fusarium* occur chiefly on open soils, and are not usually prevalent in clay, is of a similar nature. Other forms, such as *Agaricus melleus*, can thrive with relatively little air.

The reaction of the media in which they are growing is known to influence the development of many of the common moulds. In a general way these grow better in slightly acid media, whereas bacteria generally prefer a neutral or slightly alkaline reaction. There are, however, many exceptions on both sides to this general rule, and relatively few parasitic fungi have been examined to determine how they are influenced by the reaction of the cell juices. Several fruit-rotting parasites are known to be tolerant to organic acids and to use them as sources of food; moreover the strong acid reaction of the host preserves such fungi as attack apples, oranges, and the like from the competition of the bacteria that usually assist in promoting the decay of vegetable substances. The enzymes of *Botrytis cinerea* have no activity on living plant tissues in alkaline solutions, and the parasitism of this species must, therefore, depend partly on the reaction of the cell juices of the host. In the allied *Sclerotinia Libertiana* it has been found that manuring with superphosphate reduces the resistance of the host, and it is suggested that this is due to an increase in the acidity of the cell juices. In the same way, the vine is not susceptible to the attack of the black rot fungus, except when its tissues are rich in organic acids (young leaves and fruit); the parasite only grows well in acid media, and sugars are rather unfavourable to it. There is some evidence too that the reaction of the soil is of importance in the root diseases caused by species of *Rosellinia*, which are said to be most severe in acid soils. On the other hand, the wilt parasites of the genus *Fusarium* are not checked by even heavy doses of lime and are evidently tolerant of alkalis, as is shown also by their behaviour in artificial culture.

The above are some of the chief conditions in the surroundings which aid or check parasites in their growth, multiplication, and spread. It will be observed that they fall naturally into two classes: those which are controllable to some extent by the practices of good cultivation and plant sanitation; and those that, being dependent on conditions of climate and the like, cannot be modified by any means at our disposal.

**VIRULENCE.**—It seems probable that when all such external conditions which influence the parasitic activity of a fungus are allowed for, there is still another factor which may affect the intensity of the attack. This is the

innate virulence of the parasite, a subject about which there is little reliable information as regards the fungi, though it is well known to be of primary importance in many bacterial diseases.

Variations in virulence between different races or strains of the same fungus seem to be manifested in the following cases.

Occasionally it has been observed that a normally saprophytic fungus assumes parasitic activities. Some instances have been referred to above (p. 43), but the total number reported is small and it is not certain that weakness of the host plant may not have played a part in assisting infection in some of them. More definite is the evidence regarding "bridging species," where a fungus that normally is unable to infect a certain host can be got to do so by cultivating it for a time on another host. Thus the black rust of wheat can be grown on barley and the spores formed on the latter will then infect oats, though direct infection from wheat to oats will not occur.

Another frequently observed fact is the great intensity of the attack of newly-introduced parasites and the subsequent gradual decrease in virulence. Thus the hollyhook rust made the cultivation of this plant almost impossible in England when it first appeared in 1873, but now often does little damage. So also the oak mildew was very virulent when it first reached Europe about 1907, but in the last few years is said to be waning in France and Austria; and the carnation rust did great damage in the United States on its first introduction in 1890, but began to decrease about 1896.

In India, coffee leaf disease seems to have lost a good deal of its virulence since the bad years that followed its first appearance in 1869. It is difficult in these cases to separate the factors which alter the resistance of the host plant from those that affect the parasite, but it seems probable that alterations in the innate virulence of the latter are at least sometimes responsible for the changes observed. A case in point is the behaviour of the coffee leaf disease on some of the resistant varieties of coffee imported from Africa to the East. When Liberian coffee was first introduced to Java, it suffered little from the disease, but it is now attacked with approximately the same severity as Arabian. So also robusta coffee is now sometimes scarcely less injured than the older varieties, though it gained a great reputation as a disease-resister when first grown in 1901. It has been held that this is a good case of a parasite getting gradually accustomed to a new host, though on the evidence as yet available it is scarcely

safe to exclude the possibility that a gradual loss of the resisting powers of the host under new surroundings sufficiently accounts for the facts. Nevertheless it is probable that fungi must possess the power of gradual adaptation to a host that at first is relatively resistant. No other explanation seems sufficiently to account for the existence of the specialised races of certain parasites, each confined to one or a few of the hosts of the species. It is supposed that through long growth on a particular variety of host, the fungus becomes so accustomed to one set of conditions that it loses its power of living under different conditions. When forced to grow on a less congenial variety, it again gradually becomes adapted to its new conditions. Thus black rust kept confined to the relatively resistant einkorn wheat for a number of generations has been found to become progressively more vigorous, while at the same time losing to some extent its power of infecting the common wheat from which it was taken. Hence specialisation of parasitism is apparently an adaptation acquired by fungi in order to overcome the natural resistance to attack possessed by certain species and varieties of the host; and the factors which limit each form to its special hosts must be essentially the same as those which influence the predisposition or immunity of plants to disease.

CONDITIONS AFFECTING THE HOST: PREDISPOSITION: IMMUNITY.—Individual plants, like man, vary greatly in their resistance to disease; some seem to be specially predisposed to attack, others are relatively immune. It is not yet possible, however, to get a clear idea of the causes which produce this variation in the power of resisting a disease; the factors are exceedingly complex and their interaction is so close that it is often difficult to determine the effect of any single one.

Resistance to one disease does not necessarily imply resistance to another. Thus potatoes resistant to early blight (*Alternaria Solani*) may be very susceptible to Phytophthora. A variety of flax that is highly resistant to wilt may be severely attacked by rust. Rivers cotton in the United States resists wilt but not bacterial blight. American Club wheat is relatively immune to yellow rust but not to black at Cambridge, and Bobs wheat is not injured by the latter but gets orange rust in some parts of South Africa. Even the resistance of one part of a plant may not be the same as that of another. The age of the part attacked may have a considerable influence. So also the foliage and the tubers of a potato may have different powers of resistance to blight, and the leaves and fruit of the grape vine to mildew. Hence we have evidently to do with a character which must depend on subtle variations in the structure or

composition of the host, and the difficulty of investigating these variations will be apparent.

It is useful to distinguish between disease avoidance, disease endurance, and true disease resistance.

Plants often avoid disease not because they are more resistant but merely because they ripen too early or in some other way are in a condition to escape injury at the time when the disease usually develops. The early maturing varieties of groundnuts introduced into cultivation in India may suffer little from the tikka disease because their nuts are already formed before serious injury to the leaves occurs. Similarly, early potatoes may escape blight in years when the late crop is ruined, because the disease often does not begin to spread until the latter is ripening. Late peas are so severely attacked by the powdery mildew in some places that their successful cultivation is impossible. In these cases, experiments have shown that the early crop is not in any way immune when exposed to infection. In the same way, early or late sowing may enable a plant to escape a disease that attacks in the seedling stage, as in some cereal smuts and *Helminthosporium* on barley, where the temperature limits of germination of host and parasite are different, and in cabbage wilt, where infection has been found in the United States only to take place when the soil temperature exceeds 17°C.

Plants may endure disease owing to a high degree of vigour. Weakly plants naturally suffer more than those that are luxuriant in their growth, when exposed to the same amount of infection. This is well marked in coffee leaf disease, where the first invasion in Ceylon occurred on some of the best young plantations in the island and the fungus actually grows better on strong leaves, rich in nutriment, than on those of weakly bushes. Yet the damage caused is so much more in the latter case, that the disease is habitually fought by increasing the vigour of the bushes by heavy manuring and good cultivation. The stronger the plant, the better it can cope with the drain on its resources caused by the loss of leaf, and form new leaves to replace those destroyed. Sometimes the success of the more vigorous plants in resisting injury is due to the more rapid reaction of their tissues so as to check the extension of the parasite. The protective reactions made use of, particularly the formation of cork around the invaded part, have been already described (p. 100); and in such a case as the red rust of tea, the rapidity with which a strong bush can undergo these changes is undoubtedly one of the chief reasons why the disease is only severe on poor gardens. Nevertheless the argument must

not be pushed too far, for there are many cases, particularly with rapidly acting general parasites such as *Phytophthora infestans* or *Pythium de Baryanum*, where the intensity of the attack bears no relation to the vigour of the host.

In the above cases, the amount of infection may be the same on weakly as on strong plants, though the results may be very different. In another group in which variations in strength or weakness of the host affect the result, there is an actual difference in the number of infections that may succeed, in individuals of varying vigour. In these cases, there is a variation in the true disease resistance of the host and not merely in its ~~resistance~~ <sup>ability</sup> of the attack. The whole group of weak parasites can ~~usually~~ only infect hosts whose vitality is low, and when in contact with strong individuals they may fail absolutely to effect an entry. In other cases, the subsequent development of the parasite within the tissues may be stimulated by lowering the vitality of the host, as in the generalised attacks of maize smut on plants which have been exposed to ether vapour. So also it has been observed that epidemics of red rot and rind disease of sugarcane often follow severe attacks of insects such as cane fly, and it can easily be noticed that the total number of infections which develop in such cases is much greater than on canes not weakened by these pests. There is reason to believe also that the injurious effect of factory smoke may lower the resistance of plants to certain diseases, such as oak mildew in Europe. Opposed to these are the cases where successful infection only occurs on healthy individuals. They are fewer in number, but are well marked in some of the downy mildews, in white rust of crucifers, in some Erysiphaceæ as *E. Cichoracearum*, and in infection with the uredospores of many rusts. In the rusts too, it has been found that the development of the uredo stage, usually the most important for rapid spread, is interfered with as the vitality of the host dwindles; its vigour is directly proportionate to the vigour of the host.

Amongst the circumstances which may affect the general vigour of a plant, and so render it more or less liable to certain diseases as just described, there are many that depend on external conditions which can be controlled by methods of cultivation, manuring, and the like. It is unnecessary to enter here on the importance of tillage, drainage, spacing, and other practices of good agriculture in promoting luxuriant growth. It is sufficient to repeat that certain classes of disease (but by no means all) can be modified in their severity by securing a luxuriant development, both of the roots and of the above-ground parts of the crop.



Another class of external circumstances that may affect the true resistance of plants to disease is the climatic and soil conditions to which they are exposed. Why these so act is not usually known, but there are several observations which indicate how great their influence sometimes may be. It has been found, for instance, that alder trees suffer more severely, in Germany, from the wound parasite, *Valsa oxystoma*, when growing in meadow land than in their natural habitat in swampy ground, though the reason for this is not known. Larch canker is worst on calcareous soil and is rare on siliceous. The dryer side of the island of Réunion grows arabica coffee well where the soil is deep, but in shallow soil it gets severely attacked by leaf disease, and this difference in the intensity of the attack may be noticed in bushes only a few yards apart. In India, heavy soils, liable to water-logging, tend to predispose to red rot of sugarcane, though the plants may show the greatest luxuriance of growth; this seems to be due not to increased activity of the fungus but to infection at the nodes occurring more readily than in cane grown on higher land.

With regard to climatic influences, it is well known that unacclimatised plants are often more severely attacked than those that have become acclimatised in a new locality, though their growth may otherwise be satisfactory. So also the resistance of the host may be lowered by temporary conditions of the weather, though it is often hard in these cases to exclude the influence of weather changes on the parasite: in other words, it may be that what has been sometimes regarded as increased susceptibility of the host is really nothing more than the development of conditions favourable to the growth or reproduction of the parasite. Thus it has been observed that black rot of the grape is more abundant when humidity has caused a vigorous, sappy growth of the vines. Potatoes are also said to be more susceptible to blight when the leaves are turgid, a condition often depending on climatic changes such as cloudy weather. Excessive shade may produce the same result by checking transpiration, but in this, as in many similar cases, the infective activity of the parasite may also be stimulated by similar conditions and may be the chief factor in increasing the disease. This is well shown in the black thread disease of rubber, where too close planting increases the attack by giving conditions favourable to the reproduction of the fungus but does not in any way alter the susceptibility of the tapped surface. In the same way, the effect of a fall of temperature in starting the *Phytophthora* blight of potatoes has been proved to be due to increased germination of the sporangia and not to any change in the susceptibility

of the host, while the influence of dry weather at flowering time on the loose smut of wheat is to increase the disease simply because the glumes open more widely and expose the stigma to infection. Frost has been found to predispose to diseases such as pear scab and various cankers of the woody parts of trees, but the action is probably similar to wounding in that it allows the parasite to enter by destroying the outer protective tissues of the host and exposing the inner layers to attack.

It was at one time believed that varieties which possessed inheritable powers of resisting specific diseases, could retain these powers irrespective of the conditions, climatic and other, under which they were grown. Of late, a good deal of evidence has become available to show that this is not so, and that resistance may be broken down by alterations in the surroundings. Thus the wilt-resistant hybrid watermelon known as the Conqueror has preserved its character in the eastern United States but lost it when transferred to the Pacific Coast. So also several wheat varieties have been found to become badly attacked by rust at the famous Svalöf Station in Sweden, though almost immune under other climatic conditions. One of the pure line selection wheats from this station imported on two occasions direct to France, died out in a couple of generations from sterility caused by severe attacks of black rust, whereas the same pure line variety, imported after growth for a time at Hohenheim in Germany, survived. In South Africa too, the Australian rust-resisting wheat, Bobs, remained exceptionally immune to black rust at Pretoria, but in the low country and elsewhere was badly attacked. Australian rust-resisting wheats have also been found to become freely rusted in India, but as the variety of rust concerned was not recorded and they were exposed to a rust (*Puccinia glumarum*) not found in Australia, it is possible that their resistance was not really tested. Einkorn wheat, which resists all three rusts, remained resistant when imported from England to Lyallpur until killed by heat in May, but at Pusa, though resistant up to April, it became badly attacked by black rust in May and June. In South Africa, another similar case has been observed, barley remaining free from black rust in winter, even though grown alongside badly rusted wheat and known to be mildly susceptible to inoculation from wheat under normal conditions; whereas in summer it is frequently attacked.

The true resistance of a plant, or part of a plant, when exposed to infection, must depend, so far as is known, on anatomical or on physiological conditions of the tissues. In most of the cases given above, there

is nothing to show whether the factors act chiefly by causing changes in structure or chiefly by physiological alterations in the cells. In other cases, however, it is possible to form an idea of the relative importance of the two sets of conditions, anatomical and physiological, though the boundary line between them is by no means sharp.

The anatomical characters which may influence susceptibility have been in part considered in the section on infection (p. 72), where it was shown that the state of the surface layers, presence or absence of hairs and stomata, thickness of the external walls, early or delayed formation of cork, and the like, may play an important part in aiding or hindering the penetration of the germ-tube. The age of the part attacked may influence the readiness with which penetration occurs, owing to the alterations in the structure of the outer layers as the tissues mature. This is especially noticeable in the case of parasites that attack stems and roots, as the red rust of tea and the root diseases caused by *Rosellinia* and *Ustilina*, where the amount of cork formed may determine whether an infection succeeds or fails. The same is probably the reason why larch canker has been found to attack young trees from seven to fifteen years old most readily. Growth in particular soils may possibly cause anatomical conditions that influence infection. Thus the amount of bloom on barley leaves may be increased in alkali soils, and this may reduce rust owing to water running off such leaves. The variation in structural characters found in different varieties of the host plant may be important also. Thus the more waxy varieties of raspberries are those least damaged by *Coniothyrium*. Similarly, potatoes with small, hairy leaves and an open habit of growth, dry quickly after wetting and are hence less liable to blight. In some pears there is an open channel from the calyx to the carpels, and these are particularly prone to the disease caused by *Fusarium putrefaciens*. Wheat varieties with very open glumes are said to be especially liable to loose smut. Moisture may also affect the protective parts of the plant, a humid atmosphere leading to fewer hairs, a thinner and later-formed cuticle, and relatively less mechanical tissues and more parenchyma. The resistance of certain varieties of plums to brown rot (*Sclerotinia cinerea*) has been shown to be in part due to the plugging of the stomatal openings with masses of parenchymatous cells; in the less resistant kinds, only a few layers of flattened cells are formed below the stomata and penetration through the latter can occur. In this case, there is a further power of resisting attack after penetration, which seems to depend on the chemical composition of the middle lamella of the cell walls. In the resistant sorts, the hyphae are not readily able to dissolve

a passage between the cells. Possibly also the slow growth through the tissues allows of the accumulation of toxins formed by the fungus itself and capable of inhibiting its growth. As the plums ripen, susceptibility may increase, owing to a natural softening of the middle lamella so that the hyphæ can rapidly extend between the cells; this is found chiefly in the softer varieties and consequently the disease does most injury to soft-fruited kinds. Hence susceptibility to brown rot depends on several different factors, and the same is probably true for many cases of the kind. Another instance of the effect of anatomical structure on disease resistance is found in some of the cereal smuts. Each stage in the infection is dependent on the age and structure of the tissues, and even after the young shoot has been entered, the mycelium may be checked by the barrier caused by the differentiation of the vascular tissues at the primary node; only those hyphæ that penetrate this barrier before differentiation has progressed far, can reach the growing point and continue their development. Hence varieties that pass through the early stages of growth rapidly are relatively immune.

Many of the protective reactions made use of by plants in checking the invasion of a parasite are manifested by alterations in the minute anatomy of the tissues, but evidently must have their origin in physiological stimulation of the living substance of the cells and so do not properly come under the present head. In the same way the predisposition to certain diseases caused by etiolation (as in lettuces grown under frames in regard to mildew) is essentially physiological; the thin cell walls of such plants are due to disturbances in metabolism, and such disturbances are further shown by the lesser amount of sugar, protein, and volatile oils found in shaded plants as compared with those grown in full light. The more closely such cases are examined, the more the physiological factors are seen to predominate over the anatomical.

Physiological conditions in the tissues of the host plant are without doubt the most important (as they are certainly the least known) of all the factors that influence predisposition or immunity. They may sometimes be distinguished from anatomical conditions in that whereas the latter usually act by influencing penetration or growth of the hyphæ in a more or less mechanical fashion, the physiological powers of resistance frequently do not affect penetration but result in the subsequent death or inhibition of growth of the hyphæ.

Several cases have been already referred to where parasites can penetrate the wrong host plant but fail to become established. Barley

is not injured by mildew from wheat, though when conidia from the latter are germinated on a barley leaf, a haustorium is formed in the usual way. Subsequent growth is apparently impossible, because the fungus does not get the right kind of food. So also *Venturia inaequalis* from apple will send infection hyphae into the cuticle of the pear, and *V. pirina* from pear into that of the apple, but the mycelial growth is scanty and soon dies off. In the rusts, it has been found that the germ-tubes from the uredospores can enter the stomata of the most diverse plants, but further growth only takes place on the hosts proper to the species.

The behaviour of immune or resistant varieties of the proper host has also been investigated in some of the rusts. In the brome grass rust, it has been established that liability to infection is altogether independent of the structural characters of the variety. The thickness of the cell wall, presence or absence of bloom on the leaf, number of stomata, hairs, and so on, have nothing to do with infection, which depends entirely on the nature of the cell contents. In several cases (as in cereals resistant to black and yellow rusts, and chrysanthemums resistant to *Puccinia Chrysanthemi*), entry through the stomata takes place in the usual manner, but the infection hyphae form few or no haustoria. The cells of the host that surround the infection hyphae are killed, and as the fungus can only grow on living plant tissues, it is checked by the barrier of dead cells around it and soon dies. Certain varieties of apples and pears have also been observed to suffer little damage from the parasites that cause scab (*Venturia*) for the same reason, the too rapid death of the cells preventing the mycelium (which requires living food) from further growth. Hence immunity may really be due, in some cases, to hyper-sensitiveness of the living part (protoplasm) of the cells.

In many cases, physiological resistance to disease seems to be connected with the composition of the cell sap. Organic acids, tannins, and various other bodies play a part. In black rot of grapes, the readiness with which the leaves and fruit are infected at different ages has been correlated with the amount of organic acid (tartaric) in the plant juices, diminishing as the acid content falls. Those varieties that are rich in acid and form sugars late, as the Folle-blanche, are less resistant than those, like the Clairette, which have little acid and early become rich in sugar. On the other hand, wheats resistant to rust have been reported in some cases to have a higher percentage of acid than the susceptible kinds, and the quantity of acid as well as the resistance to rust are said to increase with the altitude at which the wheat is grown. In certain fruits

and green parts of plants, it has been shown that the cell sap contains an oxidising enzyme, capable of acting on substances such as gallic acid to form a tannin-like body which is toxic to fungi. Any injury, such as those that result from fungus attack, can start the reaction. As ripening progresses, the amount of this oxidase diminishes and the parts become more susceptible to attack. This is held to explain the injury caused to mature fruit by various ripe rots that do little harm to those not yet ripe. In other cases, it appears probable that the sugar content of the cells is of importance. Thus the fungus that causes red rot of sugarcane is often present in the base of the plant from an early period of growth, but remains in a more or less dormant condition until the sugar content of the cane reaches a certain point. From about the sixth month onwards, the fungus progressively becomes more active, until the crop approaches full ripeness. In mildew-resisting varieties of the grape vine, it has been noticed that the leaves lose their resistance in the autumn at the moment when food reserves are being stored up in the neighbourhood of the buds, but the changes that produce this result do not seem to have been examined.

The influence of nutrition on physiological resistance is sometimes very marked. It has been studied chiefly in connection with the action of various fertilisers on the intensity of certain diseases.

An excess of nitrogenous nutrition has long been known to predispose to some diseases. This is most marked with the rapidly assimilated nitrates. It has been found in France that a heavy dressing of nitrate of soda is sometimes sufficient, by itself, to lead to the development of grey rot in grapes, a disease caused by a universally distributed but rather weak parasite, *Botrytis cinerea*. Gooseberries become virulently attacked by mildew on the sappy growth that results from copious applications of farm-yard manure. In potatoes, the injurious effect of too much nitrate in predisposing to *Phytophthora* is well known. In this case, experiments suggest that resistant varieties owe their resistance more to internal tissue conditions than to any differences in penetrability of the outer layers, but that neither the reaction of the cell juices nor the amount of tannin present has any connection with the property. Some observations suggest that the more starchy varieties, such as Richter's *Imperator*, are more resistant than those that are rich in nitrogenous matter. In connection with wheat rust also there are many observations showing the effect of nitrogen in predisposing to the disease. Thus at Woburn in England, the nitrogenous plots in the manurial series (which has been

in existence for over thirty years) have been observed to be more rusted than the other plots in the series. In some of the former, e.g., in that which gets only nitrate of soda, the growth is now far from luxuriant, so that humidity within the crop, owing to dense stand, cannot play an important part. Wheat and barley mildews in this series are also worst on the nitrogen plots, especially that with nitrate of soda. At Rothamsted also, the high nitrogen plots are the most susceptible to several diseases (including rusts and *Epichloe typhina*), and the normally manured plots may remain quite free, though they are in close proximity and equally exposed to infection. In water cultures the same results have been obtained with both rust and mildew, as the solutions in which the usual quantity of nitrogen required for healthy growth was doubled or quadrupled, gave the worst attacks, and this occurred whether the nitrogen was given as sodium nitrate or as ammonium sulphate. A similar result has been noticed in tea manurial trials in India, where the amount of the die back disease (p. 450) varied directly with the amount of nitrate of soda applied. Though it has been found that excess of nitrogen leads to soft tissues with thin walls and a thin cuticle, it is probable that alterations in the composition of the cell sap are more important in producing increased susceptibility in these cases. *Uromyces Betae*, for instance, will grow freely on the bruised surface of mangold leaves grown with excess of nitrogen, but makes no headway when sown on a similarly bruised surface of the leaf of a normally manured plant.

Phosphates are usually believed to increase resistance to disease, but a few cases have been observed where they have the opposite effect. Thus a dressing with superphosphate has been found to reduce resistance to *Sclerotinia Libertiana*, possibly by increasing the acidity of the cell juices since the enzymic activity of allied parasites is known to depend on the acidity of the solution. In the lettuce mildew also, phosphates have been found to diminish the resistance of plants grown in water culture.

Salts of potash have a considerable reputation as essential to the maintenance of the natural powers of disease resistance in plants. Thus at Rothamsted, the potash-starved plots are always the first to succumb to parasites, such as wheat and mangold rusts, in bad years of disease. Furthermore, potash has been proved in certain cases to increase disease resistance. In water cultures in which the amount of potash necessary for satisfactory growth was doubled or quadrupled, a reduction in the amount of wheat rust and mildew has been obtained, though this effect

was not sufficient to neutralise the predisposing action of an excess of nitrogen. Similarly, growers of tomatoes under glass have found that the ravages of various fungi can be checked by potash manuring, and there are several other cases where the use of fertilisers such as kainit has been found to have a beneficial effect.

Lime has sometimes been found to increase liability to disease. The condition known as chlorosis may be induced by an excess of lime, and chlorotic plants are known to be more liable to attack by certain parasites, as *Septoria piricola* on pear. Larch canker and the wheat disease caused by *Melanospora damnosus* are also more severe on calcareous soils. On the other hand, those varieties of irises that prefer a limestone soil, are liable to be severely attacked by *Heterosporium gracile* when grown in soil deficient in that substance.

Age may have a physiological as well as anatomical bearing on liability to disease, as is indicated by cases where growth on the older parts is feeble, even after infection has been successfully accomplished. Thus the cucurbit mildew, *Erysiphe Cichoracearum*, from *Cucurbita maxima*, readily infects the cotyledons of *Sicyos angulatus*, but gives at most a feeble growth on the older leaves. So also wheat mildew cannot infect unwounded mature leaves of *Hordeum sylvaticum*, but grows readily on young leaves, or on wounded old leaves when inoculated on the uninjured epidermis near the wound. As the fungus does not penetrate beyond the epidermal cells, the loss of resistance must be physiological in this case. Many other instances are known in which only young parts are normally attacked, but as a rule it is not clear whether this is due to anatomical or physiological causes. The effect of ether and heat in allowing generalised attacks of maize smut suggests that the resistance of older parts in this case is largely physiological. Somewhat similar are the cases where infection only occurs on galls on the host plant, as in *Rosa arkansana*, which has been observed to be immune to the rose mildew, except where the leaves bore galls probably caused by a gall-fly.

These last cases lead to others where the resistance of host plants normally immune can be lowered by injuries, the application of narcotics, and the like. The mere growth of a plant under glass may reduce its resistance. Several varieties of wheat are much less resistant to black rust when grown in green-houses than in the open, and the same is true of some other rusts and mildews. The immunity of barley to the wheat mildew can be destroyed in a number of ways, as by removing a small piece of the tissues, pricking with a pin, exposing to slug bites, crushing



by pressure, immersing in warm water, touching with a hot knife, or exposing to the vapour of ether, chloroform, or alcohol. Similar results have been obtained with the mildew of *Euonymus japonicus* when tried on the immune species *E. nanus*. With the cereal rusts too, the use of anæsthetics has been found to have some effect in lowering the resistance of forms that are normally immune; black rust of barley has been got to attack oats in this way. Wounds seem, however, to have little effect in increasing susceptibility to cereal rusts. It has been conclusively shown that these results do not depend in any way on the removal of physical barriers or on any anatomical or structural injury. The loss of resistance is a physiological change, due to some internal alterations in the protoplasm or cell sap.

From the above considerations of the relation between host and parasite, it will be evident that there are various ways by which a plant may avoid being seriously damaged by a parasite. It may escape injury by the more or less accidental circumstances that it ripens too early or too late to give the parasite the best chances for successful attack, or that the weather conditions at susceptible periods of its life are not suitable for infection. It may endure disease and give a satisfactory yield even when attacked, owing to the strength and luxuriance of its growth. It may finally resist disease, owing to some—as yet little understood—properties of its tissues, especially, as it seems, of the living protoplasm and the cell juices.

Each of these three ways of escaping injury may be (and is in practice) made use of in the fight against plant diseases. Early groundnuts are grown to avoid tikka disease; coffee and tea are made as luxuriant as possible by manuring, drainage, and good cultivation, to diminish loss from leaf disease and red rust; resistant varieties of potatoes are introduced where blight is prevalent. In this last direction the work is as yet in its infancy where field crops are concerned; but in some countries, notably the United States, considerable progress has already been made in the selection and breeding of resistant varieties of crops.

#### SELECTION AND BREEDING OF RESISTANT VARIETIES.—

Many diseases are not easily checked by any direct method of treatment at present practicable. Such are the rusts of cereals and other field crops, and the wilts caused by *Fusarium* and allied fungi. In these cases, use has been made of the observed differences in susceptibility possessed by different varieties of the host plant. Every degree of resistance may occur, from total immunity, such

as is possessed by Buri cotton to wilt or by European flax to the race of flax rust found on Indian linseed at Pusa, to temporary resistance, such as occurs in some thick varieties of sugarcane in regard to red rot. It has been found that the character of resistance to a specific disease may be inherited, and that in some cases it follows the laws of inheritance first outlined by Mendel. This has made it possible by methods of breeding (which it is unnecessary to enter on here) to combine by hybridisation the resistant quality of one variety with other desirable characters, such as productiveness, of another variety. A certain number of resistant hybrids has thus been introduced into cultivation in recent years.

Furthermore, it has been found in a good many cases that mere selection of individually resistant plants, without crossing, has given a strain that maintains its power of resisting disease often for a considerable number of years. Thus several wilt-resisting varieties of cotton have been introduced by the United States Department of Agriculture, by selecting individually resistant plants and growing the best of them pure for several generations, until it was demonstrated that the character was definitely inherited. The work has been carried on since about 1900, and the results are remarkably satisfactory in that the area under these cottons in wilt-infested tracts is now considerable. Deterioration occurs if selection is neglected for long, owing to admixture of seed, cross-pollination with susceptible kinds, and variation or reversion within the variety. In the same way, rust-resistant wheats are being produced in the United States and elsewhere. Other successful instances are the *Fusarium*-resisting cowpeas, watermelons, flax, and cabbages, rust-resisting asparagus, and anthracnose-resisting beans and clover. The production of blight-resisting varieties of potatoes in England and elsewhere is mainly in the hands of specialists, and the new kinds are continually being introduced into cultivation. They are obtained chiefly by selection from plants grown from the seed, not the tubers. Recently, varieties that resist warty disease have become available. In India, variation in disease resistance has been observed in several crops, and the wilt-immune Buri cotton has been introduced with much success into areas where the disease is severe. With sugarcane too, considerable temporary improvement has been effected by replacing varieties susceptible to red rot by others which resist the disease for a time. In this case, the disease is usually only severe in the thick varieties of cane chiefly grown in the tropical parts of India; the thin canes of the north are much less susceptible. Thick canes are often grown under conditions which predispose to

red rot, as in heavy, poorly-drained soils, and frequently die out from the disease after a few years. It has been found necessary in certain areas to introduce healthy varieties from other parts of India at intervals, and no cane has as yet been found which will stand the local conditions of cultivation in such places as the Godavari delta for long. On the other hand, many of the thin canes are highly resistant, and it is believed that it may be possible to introduce this character into thick varieties by crossing, as has been successfully accomplished in Java. It is also possible that by growing canes from seed instead of from setts, new varieties of thick canes, more resistant to red rot than those at present in cultivation, may be obtained by selection, as experience in Java and the West Indies indicates that such seedling canes often possess disease-resistant qualities to a high degree.

Occasionally it is possible with woody plants to secure a healthy crop by grafting or budding non-resistant kinds on resistant stocks. The classical instance of this is the saving of the French vineyards from destruction by *Phylloxera* through the use of American resistant stocks. The same method is being used in India against the Khasi Hills orange disease, the cause of which is obscure.

**DETERIORATION.**—There is a good deal of evidence that plants may become progressively more susceptible to disease under certain conditions. Two circumstances seem to be especially concerned in this change: habitual propagation from vegetative parts not from seed, as in potatoes and sugarcane; and growth under conditions of climate and soil other than those found in the natural habitat of the plant, as in the cultivation of exotics.

With potatoes, it is a common experience that few varieties last for many years in Europe without becoming susceptible to potato blight. In the production of new varieties, resistance to blight is one of the primary aims, but it is rare to find a sort that can maintain its resistance for many years. The more resistant kinds may be disease-free for the first few years, but then usually begin to be attacked and few last much beyond 20 years. Varieties raised from self-fertilised seed deteriorate, as a rule, far more easily than those that result from cross-pollination. Deterioration may often be postponed by change from one district to another. Tubers grown in Scotland and used for seed in England and Ireland have been found to suffer less from disease than the same variety grown continuously in the one district. With sugarcane too, something similar has been observed in India. The introduction of canes for planting from

other localities has been found, as mentioned above, to diminish loss from red rot. Even after a variety has become highly susceptible through continuous growth in a particular area, fresh sets of the same variety obtained from another district may give a healthy crop. In Java, it has become a regular practice to grow the seed crop in areas other than those in which the field crop is grown, in order to check the ravages of the obscure disease known as "sereh."

Evidences of deterioration are also sometimes found in crops grown from seed. Most of the cases reported refer to exotics, which in their natural home are exposed to a different environment. Groundnuts seem to become progressively more subject to the tikka disease by cultivation in India, and the last great outbreak of this disease in the Bombay Presidency about 1900-02 was effectively dealt with by the introduction of seed from Madras, Japan, and the United States. There are indications from the past history of the crop in India that varieties grown too long in the one district become gradually more liable to tikka disease, and that change of seed is the best way to check the loss. Another similar case is the obscure disease of rice known as "brusone" in southern Europe. It has been found that this disease can be successfully checked by the use of seed from localities with different environmental conditions. At first seed from distant countries, chiefly from Japan, was used, but of late it has been found sufficient to exchange seed between different localities in the same country.

In several of these cases, it has been found that deterioration may be postponed by good cultivation. On the Government Farms in India, both sugarcane and groundnut have been found to maintain their health longer than under the often defective agricultural practices of the ordinary cultivators. So also deterioration may be accelerated by neglect, and disease resistance in potatoes and sugarcane is soon lost through bad cultivation.

The factors concerned in these cases are unknown, except that they are in the host not in the parasite. That plants propagated vegetatively may become worn out through age, and hence more subject to disease, is probable. That "change of air" may have a beneficial effect in checking deterioration is clear from our own experience. That organisms, living for many generations in countries in which they may not become perfectly adapted to the climate and mode of life, degenerate, is also not opposed to experience with man and the higher animals. But what the exact factors are that cause these changes, and how they are acted on by the

external conditions is not known, and attempts to check deterioration are hampered by this want of knowledge.

**EPIDEMICS.**—An epidemic may result from the action of a single factor or from a combination of several. In many cases, the mere introduction of the parasite into a new area, where the host exists, is sufficient, and some of the most disastrous outbreaks of epidemic disease in plants have arisen in this way (vine diseases in Europe, chestnut blight in the United States, blister blight of tea in Darjeeling). In others, as in wheat rust, the occurrence of particular climatic conditions at a certain season may cause a generalised attack. It is probably safe to say that every year is potentially a year of severe rust in the wheat crop in India. The parasite is sufficiently widely distributed throughout the fields to give rise to an epidemic outbreak if weather conditions become suitable. In the United Provinces and western Bihar, the deciding factor is usually the humidity of the air during the months of January and February. An analysis of the conditions in three selected districts, Allahabad, Jhansi, and Benares, during the years 1890—1903, gave the following results. In Allahabad, the four seasons in which humidity was most largely in excess in January and February taken together were the four most rusty years. Similarly in Jhansi, the three dampest seasons in these months were the seasons of worst rust. In Benares, the five years with most moisture in January and February combined include the four years of worst rust, while if January alone be taken (the harvest being earlier in this district than in the others) the parallelism becomes exact.

In other cases, as in potato blight in the plains of India, several factors may combine to cause an epidemic. The disease was possibly first introduced into India in the Nilgiri Hills between 1870 and 1880. In the north, it broke out near Darjeeling in 1883, shortly after the introduction of English varieties into the district. It spread rapidly along the hills, and is now prevalent in the Himalaya and Khasi Hills wherever the crop is grown. In 1901, it was reported for the first time in the plains of Bengal, but seems to have died out after causing a good deal of damage in 1901-2. Nothing more was heard of it in the plains until 1913, when a severe epidemic occurred in Rangpur and Bhagalpur, since when again it has not been further reported. The conditions which led to the 1913 outbreak were investigated. It was found that, owing to the ravages of the potato moth, there was little seed available locally from the 1912 harvest. As a result, the price of seed potatoes rose very high and a large import took place from the Himalaya. The temperature in the

plains at the end of the 1912 monsoon was unusually low, especially at night, and as the new seed was, in part, got down late, it was not exposed to much heat before sowing in October-November. The disease is endemic in the localities from which the seed was procured and doubtless many of the tubers contained living mycelium of the parasite. In the ordinary course of events, such mycelium would have been killed by the heat in the plains (as it is known to be unable to survive prolonged temperatures in the neighbourhood of 90°F.), but it is probable that imports continued after the cool weather had set in and that living mycelium was planted with the crop. Conditions favourable to the spread of the disease appeared in the latter half of December, when there was much fog and cloud, and by January an epidemic was in progress. Attempts to carry the fungus over through the hot weather and rains failed at Pusa, and the freedom of the crop in the infected area the following year indicated that, under natural conditions, it was unable to remain alive from one season to another. Hence the outbreak in the Gangetic Plain in 1913 is to be attributed to the action of several distinct factors: the planting of a large amount of hill seed likely to be from a diseased crop; the exposure of the tubers to a lower temperature than usual in the plains before planting; and the occurrence of favourable conditions for the spread of the disease as the crop ripened. Any one of these alone would probably have failed to produce an epidemic.

#### Methods of direct control.

THE use of poisons or other agencies, such as heat, to kill parasitic fungi or prevent their spores from infecting healthy plants, is common in some countries and with certain crops. It is capable of only limited application in India under present conditions, for various reasons. In the first place, it requires to be carried out with certain precautions: to be done at the right moment; to be repeated if sudden rain or other accidents are likely to have interfered with the results; to be applied to the right parts of plants, and so on. It is just in matters of this kind that any community in which the general level of education is low, as amongst cultivators in India, is liable to fail. Secondly, it is often rather expensive, and the crop must be of a valuable nature to repay the cost. Hence it is more applicable to vines, orchard and market-garden cultivation, planters' crops, and very heavy field crops, than to the ordinary run of Indian crops. However there are certain cases in which it can profitably be employed, even in India. Amongst these are field crops such as potato and possibly groundnut, where the produce is valuable and

in which there is a liability to total or very great destruction from disease. It is also practicable in market gardens and in small plots such as are often found in villages, where as a result of manuring, much heavier outturns are obtained than in ordinary field cultivation. Finally, it is applicable to fruits, to valuable garden produce, and to planters' crops such as tea and coffee, in which the profits may be sufficiently large and the advantages of intensive cultivation sufficiently marked to permit of a considerable increase in "cultivation" expenses with a view to obtaining the maximum yield from a given area.

There are two main classes of parasites which can be usefully attacked by poisons ("fungicides"). In the first place, ectophytic parasites, passing as they do the greater part of their lives on the surface of the attacked plants, may easily be reached by the superficial application of a poison. In the second place, those endophytic parasites which are disseminated by spores that fall on the surfaces and thus infect healthy plants, may be attacked during or before the sporiferous stage and their spores killed, or the exposed surfaces may be coated with a substance which inhibits infection.

To the first class belong the conidial stages of the Erysiphaceæ or powdery mildews, which live on the surfaces of the leaves of many plants, usually sending only haustoria into the epidermal cells. All the mycelium except the haustoria is superficial, and the spores are produced directly on the external hyphæ. A spray of some poisonous substance in solution, or the gas given off by powdered sulphur dusted on the leaves, is easily able to reach and kill the parasite.

In the second class are a large number of fungi which live within the plant and whose mycelium is therefore out of the reach of superficial poisons. These are, however, obliged to come to the surface to reproduce, and their spores are produced on hyphæ exposed to the air. They are, therefore, vulnerable at this stage, and the spores can be directly attacked and killed, or a layer of some poisonous substance deposited by spraying on healthy plants, so as to prevent the germination or kill the germ-tubes of those spores which may fall on the surface.

A fungicide must fulfil two conditions: (1) The poison employed must be capable of killing the parasite or preventing infection by it but must not be injurious to the host plant. (2) It must be easy to prepare and handle and reasonably cheap. In applying it, the following rules should be observed:—(i) The fungicide must be of good quality and well prepared,

otherwise it will not do its work efficiently ; some of the solutions require a little care in their preparation, others are frequently found on the market in an adulterated or decomposed state. (ii) It must be employed at the right season ; on warm, sunny days for the sulphur compounds ; not during the blossoming period of fruit trees so as to avoid injury to the delicate blossom ; at the time or just before the reproductive period of the fungus, when dealing with endophytes, and so on. (iii) It must reach the parts of the plant on which the fungus occurs or those parts of healthy plants through which infection takes place.

Two chief classes of fungicides are employed : powders, and fluids or fluid suspensions. The powders, of which sulphur is an instance, are dusted or blown on the plant to be treated. This is best done by special bellows devised for the purpose, or the substance can be dusted on from



FIG. 47. Sulphur dusting machines.

a tin vessel with the lid pierced with holes (like a large pepper castor) or even from a muslin bag ; these are less efficient and economical than the bellows. The fluids are sprayed on by a syringe or special spraying machines. With some of these, a spray can be thrown so fine that it hangs like a mist. Very many different types are in use and several are available in India.



Amongst the most useful sprayers are those intended to be slung on the back or shoulder and worked either by a single charge of compressed air or by continuous pumping. The compressed-air forms are made in



FIG. 48. Compressed-air sprayer in use.

various sizes, those holding from 1 to 5 gallons being most useful for ordinary work. In many parts of India, 4 gallons is about the limit that a coolie can conveniently work with. They are filled with the spray fluid, and air pumped in until the pressure necessary for complete discharge is reached. Sometimes the pump forms part of the sprayer, in other cases it is separate

and can be fitted on as required. In the battery system of charging, the pump is fixed to a large reservoir of the spray fluid, and serves to load a



FIG. 49. Continuous-pumping knapsack sprayer in use.

number of sprayers so as to keep several men constantly employed in spraying. Each sprayer is first charged with air and the fluid then pumped in to the necessary pressure, which is shown on a gauge. As fast as one is removed from the charging platform, it can be replaced by another. When empty of fluid, the air remains, and recharging with fluid is all that is required. In the continuous pumping machines, the pressure is maintained by pumping with one hand, while the spray is directed with the other. The knapsack forms carried on the back are the most convenient. The work is more severe than

with the pressure sprayers, and the attention of the operator divided between pumping and directing the spray.

Other types which are useful in certain classes of work are the bucket pumps and barrel sprayers. Bucket pumps can be used with any convenient vessel for holding the solution, such as a tub or kerosene tin. The pump has a foot-rest on an arm outside the vessel, by which it can be held in position; while the pump cylinder rests on the bottom of the vessel and is worked by a plunger in the ordinary way. Barrel sprayers are generally mounted on wheels, with the pump at the side or end. These two classes are most useful for orchard work. Large power sprayers are used in orchards and hop gardens in some countries, but there is as yet little demand for this type of machine in India.

In all cases, the spray jet is directed through a tube, part of which is flexible and may be of any convenient length and part is rigid and is held in the hand to direct the spray. The latter is formed by passage of the jet through a special nozzle, of which there are several good types. The efficiency of the treatment is largely dependent on the nozzle, which should throw a fine, hanging mist rather than a broken stream of drops. This saves fluid and secures an even deposit on the sprayed parts.

Before purchasing a machine it is always well to take expert advice, stating for what purpose the sprayer is to be used.

**Seed disinfection.**—This is employed against parasites that are carried on or in the seed and is, in some respects, the most successful of all the direct methods that have been introduced for checking disease. It is used chiefly against grain smuts, and is based on the knowledge that most of these are carried by the seed from the previous crop, either in the form of spores on the surface of the grain or as living mycelium inside the tissues. In the first case, the spores are within reach of fungicides, and the infected seed may be either steeped in a solution of the poison or the latter may be sprayed or sprinkled on the grain heap, which is turned over until all the grains are evenly wetted. Several substances are known which are sufficiently powerful to kill the spores without causing serious injury to the grain (*e.g.*, formalin, copper sulphate, potassium sulphide), and the details of the methods employed in using them are given under the diseases against which they are recommended. For those cases where the parasite occurs as living mycelium within the tissues of the seed, poisons cannot, as a rule, be made use of, because such as penetrate the tissues are injurious to the seed. Fortunately it has been found that the mycelium is less resistant to heat than the host is, and that it is possible, by exposing the seed to heat or by immersing it in warm water, to destroy the parasite without seriously affecting the resulting crop. The method is described in detail under the loose smuts of wheat and barley.

**Soil disinfection.**—Attempts to find a practicable method of killing spores and mycelium in the soil, which might be employed on a field scale at a reasonable cost, have hitherto failed. Such a method would be of value against the *Fusarium* wilts, *Rhizoctonia*, the root diseases of tea, coffee, and rubber, and several other soil-borne diseases, and tests have been made with many substances, such as formalin, sulphur, and iron sulphate, but have been either ineffective or too expensive for practical application. In seed beds and plant houses, formalin and other substances have proved of some value, but heating, either by steam or by surface firing, has generally given more satisfactory results. There is little scope for either of these methods in India as yet. Heavy applications of lime are used with success against "finger and toe" (*Plasmodiophora Brassicæ*) in other countries, but this disease has not yet been reported in India. Lime is also employed in soil infected with some of the root parasites of tea and coffee (see p. 441).

**Spray fluids.**—Very many substances have been used as fungicides in the past, and the number still employed in certain localities and under

specific conditions for certain valuable crops, such as the grape vine, is considerable. For practical purposes, however, the list may be reduced to a few that have proved applicable under very varying conditions and that have established themselves as efficient, when properly made and employed, in a large number of cases.

To be effective, the mixtures should be carefully made, according to the directions, from materials of the best quality. This is particularly important in Bordeaux mixture, where the composition and properties of the mixture vary considerably, and the dividing line between a good and a worthless preparation is narrow. The spraying should be done at the proper time, which varies with the crop and the disease, and in some cases the mixture must be freshly prepared as required.

The organisation required to spray a large area, such as a tea garden, is considerable, and separate publications are available from the scientific departments of various Planters' Associations in India, which give full details of the special features of such work.

WHEN TO SPRAY.—Some sprays are applied with a view to killing the parasite or its spores, either when in a dormant condition or during active growth. The dormant sprays are generally applied to woody plants during their period of rest, in order to kill spores or dormant mycelium on the surface of the plant, generally on the woody parts. They may safely be employed at a greater strength than when used during periods of active growth of the host. In some cases they are enough to control a disease sufficiently for practical purposes (*e.g.*, in peach leaf-curl), in others they are preliminary to further sprayings, during the active period of the host. Spraying with a view to killing the parasite during periods of active growth is only practicable, as a rule, with ectoparasites such as the Erysiphaceæ, endoparasites being out of reach of the fungicide while growing within the tissues of the host. It may be done whenever the parasite appears and continued as frequently as is necessary to check growth and reproduction.

Most sprays are, however, used to prevent infection by covering the healthy parts of the plant with a fungicidal layer. They are usually applied when the plant is in leaf, the first application at or just before the period when the disease appears, and others often enough to replace the fungicide when it gets washed off by rain, or begins to lose its effect, or when new leaves are formed that require to be similarly protected. If rain falls before the fungicide has properly dried on the leaves, say

within 12 to 24 hours, much may be lost and an early repetition may become necessary. Hence spraying should be done where possible in dry weather and as early in the day as practicable. Properly applied and well-dried Bordeaux mixture will go through a month or even six weeks of the monsoon with little loss, in favourable cases, but a fortnight or three weeks may be generally considered satisfactory. If growth be rapid, new surfaces may require to be covered every ten days or fortnight, but no general rules can be laid down. When several sprayings are required, the use of stock solutions is very convenient. The amount of solution required for each spraying varies with the crop and stage of growth, but from 80 to 150 gallons per acre are usually enough and from 100 to 120 gallons are, perhaps, most commonly required.

**COST OF SPRAYING.**—During present conditions, the price of the chemicals required for spraying is very fluctuating, and it is useless to attempt to give estimates. The total cost of spraying potatoes against blight in the Khasi Hills, excluding cost of machines, is about 12 to 13 rupees per acre, Bordeaux mixture being used and two applications of 120 gallons being given. The extra yield obtained in an average year gives a profit of about 100 per cent. on this expenditure. The cost of spraying areca palms with Bordeaux mixture and resin, to check koleroga disease, has worked out at from 8 to 10 rupees per acre for all charges except cost of machines. In this case, a single spraying, which only consumes about 40 gallons of mixture per acre, is given, and the chief item is the wages of the spraying coolies who have to climb the trees. The resulting profit has averaged about Rs. 100 per acre in Mysore.

**DANGER OF SPRAYING.**—Since many of the fungicides used are poisonous, it is natural that uneasiness has sometimes been felt in regard to their use on parts of plants intended for consumption. Numerous tests have shown that these fears are groundless. Thus, some years ago, there was a scare in New York owing to the large amount of grapes covered with Bordeaux mixture that were exposed for sale. Examinations of the worst samples obtainable, however, showed that it would be necessary to eat 3,000 lb. (including the skins) to obtain a dose of copper that might be regarded as dangerous. So also it was found in another experiment that sprayed celery, stripped and prepared for market in the usual way, could be consumed to the number of 66,400 heads before copper poisoning need be feared. With tea sprayed with Bordeaux mixture against blister blight in Darjeeling, it was found that copper could be detected in the manufactured leaf, but that one would have to

consume some 140,000 cups, as ordinarily made, before the amount of copper recognised as a dangerous daily dose would be reached. From these experiments, it is clear that the copper sprays are not likely to contaminate parts used for consumption to any appreciable extent. The other sprays are even less likely to be harmful.

**PHYSIOLOGICAL EFFECTS OF SPRAYING.**—It is now recognised that certain of the spray fluids, notably Bordeaux and Burgundy mixtures and the lime-sulphur combinations, exercise, at times, a beneficial effect on plants, apart from their fungicidal properties. This effect has been chiefly observed with Bordeaux mixture, which has been found to prolong the vitality of the green parts of certain annual plants, like the potato, and to preserve the leaves of deciduous trees, such as the apple, beyond the period at which they normally fall. In potatoes, an increase in the length of the life of as much as 25 days has been reported, and the resulting increase in yield, even when no appreciable disease has been present, is often such as amply to repay the cost of spraying. The action of Bordeaux mixture on vine leaves leads to an increase in the green colouring matter, and greater thickness, strength, and stiffness of the leaf. Such leaves remain green longer in autumn than those unsprayed and prolong the active growth-period of the plant. Young pear trees regularly sprayed have been noticed to grow much faster than those not so treated. The exact manner in which this stimulating effect is produced is not known, but one of the actions of surface films, such as that produced by Bordeaux mixture on leaves, has been proved to be a marked increase in the transpiration of water. If to this is joined an increased activity of the chlorophyll, as seems to be established, then increased assimilatory energy would naturally result, provided the supply of water and salts be maintained. Whether the absorption of small quantities of copper may exercise more direct stimulating effects on the plant (such as result from small doses of other substances not required for the nutrition of the plant, *e.g.*, boron) has not been definitely established, as it is difficult to exclude the indirect effect that copper may produce by influencing the bacterial life of the soil. At the same time, there is evidence to show that copper is actually taken into the leaves in small amount, and it seems probable that it directly stimulates the vital activities of the plant.

A similar effect has been observed to result sometimes from sulphur dusting. Sulphured vines may become greener and more vigorous than those not treated. In India, too, sulphuring for red spider on tea may result in the production of an extra flush.

**COMBINATION OF FUNGICIDES WITH INSECTICIDES.**—Some of the substances used as fungicides, notably lime-sulphur, have valuable insecticidal properties as well. In other cases, an insecticide may be added to the spray fluid without in any way diminishing the activity of the fungicide. Thus Paris green may be combined with Bordeaux mixture, at the rate of 5 oz. to every 50 gallons of the mixture. Lead arsenate and nicotine may safely be added to either Bordeaux mixture or lime-sulphur.

**BORDEAUX MIXTURE.**—This is at once the earliest, the most widely known, and the most generally useful of all spray fluids employed against parasitic fungi. Its advantages are (1) its adhesive properties, (2) its thorough effectiveness as a fungicide, (3) its relative cheapness, (4) its safety to handle, (5) its harmlessness, ordinarily, to the sprayed plant, (6) its beneficial effect on green plants apart from its fungicidal properties. Its drawbacks are (1) the necessity of taking some trouble in its preparation, (2) the liability of the copper solution to injure vessels made of iron or zinc, (3) the occasional injurious action of the mixture on leaves and fruit, as on peaches and some varieties of apples and plums, (4) the colour which is imparted to sprayed plants, making it unsuitable for ornamental plants or for fruit ready for marketing.

The formulæ used for preparing the mixture vary considerably, and it cannot be said that there is any general agreement as to the best to use. The chief strengths recommended are:—

4-4-50

Copper sulphate (bluestone)	..	..	4 lb.
Quicklime	..	..	4 lb.
Water	..	..	50 gallons.

This may be selected for general use on fruit trees (except peaches) and on most field, garden, and planters' crops.

5-5-50 or 1 per cent.

Copper sulphate	..	..	5 lb.
Quicklime	..	..	5 lb.
Water	..	..	50 gallons.

This is much used in general spraying and is often known as 1 per cent. Bordeaux, as it contains 1 per cent. by weight of copper sulphate. A 2 per cent. solution, in which the amount of copper sulphate is doubled without increasing the other ingredients, is advocated for spraying potatoes against blight in some countries, and the same, with the lime

also doubled, is used against koleroga of the areca nut in Mysore. The 2 per cent. solutions, with varying quantities of lime from equal to one-third, are still employed in some parts of Europe against vine diseases (especially black rot), but the tendency seems to be in favour of the more general use of weaker solutions.

$2\frac{1}{2}$ – $2\frac{1}{2}$ –50 or  $\frac{1}{2}$  per cent.

Copper sulphate	..	..	..	$2\frac{1}{2}$ lb.
Quicklime	..	..	..	$2\frac{1}{2}$ lb.
Water	..	..	..	50 gallons.

This weak Bordeaux is less effective as a fungicide than the 1 or 2 per cent. mixtures, but has gained favour in parts of Italy, and is also used for spraying fruit trees that are liable to injury from the stronger solutions.

*Directions for use.*—The mixture should be made up as required from the raw materials. There are various ready-made mixtures, proprietary powders, pastes, and the like, on the market, but none of them can be held to have proved equal to the home-made Bordeaux and many are practically worthless.

The raw materials should be of good quality. Copper sulphate guaranteed 98 per cent. purity should be obtained and is generally to be had without difficulty from reputable firms. Only freshly burnt stone-lime should be used; air-slaked or builders' lime will not make Bordeaux mixture. Where there is a difficulty in obtaining this, as is especially the case in many parts of India in the monsoon, Burgundy mixture should be employed. After mixing, spraying should be done at once, preferably the same day and never after more than two or three days. The mixture soon loses its efficacy on standing, and the rate of the change depends largely on the temperature, being more rapid in hot weather. At temperatures above 75°F., spraying should always be done the same day, as deterioration may set in within six hours. The colder the water used, the better the mixture.

To make the mixture, dissolve the copper sulphate in half the water, in a wooden vessel, by suspending it in a piece of gunny sacking just immersed. It dissolves slowly and may conveniently be put in over night. Then slake the lime in a second vessel with a few pints of water, added little by little, until the bubbling ceases, after which pour in the rest of the water. Then mix the two solutions together, either by pouring one into the other or the two together into a third vessel. Pour the lime



through a strainer to keep back all lumps and stir the mixture all the time while pouring. Stir and strain again when pouring into the sprayer. Some recommend dissolving the copper in nearly all the water, keeping only enough back to slake the lime and make it cool. This gives a weak solution of the copper sulphate and a strong milk of lime. These may be mixed by pouring one into the other, preferably the copper into the lime.

It has recently been pointed out that if stock solutions are used, and all the water required for dilution be added to the lime, then a galvanised iron vessel may be used for mixing. In this case, all that is necessary is to pour the copper sulphate stock solution into the middle of the diluted lime solution; the chemical change which takes place makes the copper sulphate harmless before it reaches the sides of the vessel. The latter must be thoroughly washed out after use.

It is often convenient to make up concentrated stock solutions of the two ingredients and mix them together when required. For this purpose, 50 lb. copper sulphate may be dissolved in 50 gallons water in a wooden barrel. Similarly, 50 lb. quicklime is slaked in a small quantity of water, added little by little, and more water poured in up to 50 gallons. The material used for keeping the milk of lime in need not necessarily be wood but a barrel is generally the most convenient. Each gallon of water contains 1 lb. of copper sulphate or of lime, and for use, the necessary amount is withdrawn from each barrel and diluted with water before mixing together. For instance, to prepare a one per cent. mixture, take 5 gallons of the copper sulphate solution and add 20 gallons water in a wooden vessel, then take 5 gallons from the milk of lime and similarly add 20 gallons water, finally mix the two together as described above, and the mixture is ready for use. The strong lime solutions should always be well stirred before drawing off, and poured through a strainer. Even after mixing, it is always advisable to pour through a fine sieve into the sprayer to avoid danger of clogging. The stock solutions keep well if placed under cover and the barrels closed with a well-fitting lid.

Bordeaux mixture as generally prepared is alkaline, containing an excess of lime and turning red litmus paper blue. An excess of copper compounds is dangerous to the foliage of many plants and is indicated by the solution turning blue litmus paper red. Another way of determining if the mixture contains an excess of copper, is to immerse a bright iron or steel surface, such as a knife-blade or a nail, in the upper layer of liquid, when a deposit of copper will form on the metal if the mixture is

unsafe. A more accurate test can be made with ferrocyanide of potassium, a few drops of the mixture being added to a little ferrocyanide in a saucer. If no change occurs, the mixture is safe, but if a reddish-brown colour appears, too much copper is present. More lime solution must be added if these tests indicate that copper is in excess, until the excess disappears. Some prefer to make up the mixture with no excess of lime, using only just enough of the latter to precipitate the copper—a quantity which varies with the purity of the lime, but can be determined by testing with litmus paper until neither red nor blue litmus changes colour. It is said, however, that when made up in this way, the mixture does not adhere so well, and is hence not so valuable in practice as the usual type. In other modifications, in which lime water is used instead of milk of lime and there is no excess of lime, it is claimed that the fungicidal properties develop with greater rapidity after spraying than with mixtures prepared in the usual way. It has been pointed out, however, that this is a doubtful advantage, as it is better to have a mixture which preserves its activity than one in which the fungicidal value is ephemeral. It is wiser, therefore, to adhere to the methods of preparation given above.

*Bordeaux mixture with resin.*

Washing soda	..	..	1 lb.
Resin (common resin or colophony)	..	..	2 lb.
Water	..	..	1 gallon.

Boil the water, then add the soda. When dissolved, add the resin and boil for about an hour, stirring continually.

Add this, when cool, to Bordeaux mixture prepared as above, at the rate of 1 gallon to every 24 gallons of Bordeaux.

This mixture has given very good results in the spraying of areca palms for koleroga in India, as the resin increases the adhesiveness of the Bordeaux to a marked degree. A weaker solution, containing only half as much resin, has also proved to be superior to ordinary Bordeaux in spraying potatoes in the Khasi Hills. In both these cases, the spraying has to be carried out during periods when heavy rain is frequent, and in all such cases it is advisable to use resin.

**BURGUNDY MIXTURE.**—This is sometimes called soda Bordeaux and is increasing in popularity, especially in localities where there is any difficulty in procuring freshly burnt stone-lime. It is claimed to have, in addition, some advantages over Bordeaux in that (1) it adheres longer to the plant and is less easily washed off by rain, (2) it is more persistent

to make, and (3) it is not so liable to clog the sprayer, as, if properly made, there is no sediment. On the other hand, it is slightly more expensive, as soda costs more than lime.

## 2 per cent.

Copper sulphate	..	..	10 lb.
Washing soda	..	..	12½ lb.
Water	..	..	50 gallons.

This is the standard mixture which is extensively used against potato blight in Ireland, with excellent results. A similar formula, but with varying amounts of soda from 11½ to 15 lb., has been long used for vine spraying in France.

## 1 per cent.

Copper sulphate	..	..	5 lb.
Washing soda	..	..	6½ lb.
Water	..	..	50 gallons.

Equally good results have been got in some cases when this solution is used instead of the last.

## 0·4 per cent.

Copper sulphate	..	..	2 lb.
Washing soda	..	..	2 lb.
Water	..	..	50 gallons.

As with Bordeaux mixture, the present day tendency is in the direction of employing weaker solutions, and in France and Germany the 0·4 per cent. mixture is popular in some localities, especially for spraying vines. Strictly speaking, there should be about 2·3 lb. soda in this formula, but the small excess of copper is allowed, as it is believed to hasten the fungicidal action, while not enough to cause injury to the leaves.

Make up exactly as in Bordeaux mixture, using soda instead of lime. The materials used should be guaranteed 98 per cent. purity. Test as before with blue litmus paper and add more soda solution if it reddens. Excess of soda may cause scorching, and can be detected by red litmus paper turning blue, in which case more copper sulphate solution must be used.

## AMMONIACAL SOLUTION OF COPPER CARBONATE.

Copper carbonate	..	..	5 oz.
Strong ammonia ("Liquor ammoniæ fortior")			
B. P.)	..	..	3 pints.
Water	..	..	50 gallons.

Make the copper carbonate into a thin paste by adding  $1\frac{1}{2}$  pints water. Then add the ammonia slowly, stirring all the time. This gives a deep blue, clear solution with a little undissolved copper carbonate at the bottom. Keep in a well-closed vessel until required, when make up to 50 gallons with water. The strong ammonia is dangerous to handle in a hot climate and may, therefore, be diluted with five times its volume of water as soon as obtained. Naturally five times as much of the dilute solution must be used to dissolve the copper.

The mixture is inferior to Bordeaux, and should only be used when the stain caused by the latter is objectional, as on ornamental plants and on fruits nearly ready for marketing. It leaves no mark on the plant.

**LIME-SULPHUR.**—The lime-sulphur solutions have come into prominence recently and have even shown a tendency to replace Bordeaux mixture for general use in the United States. It has been found, however, that they are not of value against some of the diseases most frequently treated by spraying, such as potato blight, and it is probable that they will ultimately find their place chiefly in orchard practice, where fruits such as peaches, plums, and apples are liable to be injured by Bordeaux, and against the powdery mildews (*Erysiphacæ*), for which sulphur is recommended, and can suitably be applied in this form. Furthermore, the lime-sulphur combination has proved useful against certain insects and allied pests (as red spider on tea), and there is an obvious advantage in using it where both fungicidal and insecticidal action is required.

*Concentrated lime-sulphur.*

50-100-50.

Quicklime	..	..	..	50 lb.
Sulphur	..	..	..	100 lb.
Water	..	..	..	50 gallons.

Place enough water in the cooking vessel (which may be of iron or zinc, but must not be of copper) to finish with 50 gallons of the solution. Bring to a boil and start agitating it while adding the lime. Then put in the sulphur and keep stirring till the lime is slaked and the sulphur well broken up. Boil the mixture for from 50 minutes to an hour (but not more) stirring from time to time. This gives an orange-red liquid, with a small sediment of lime and sulphur. Strain off through coarse gunny sacking and store so that air cannot reach the solution, as in well-closed iron drums or in closed barrels to which a layer of mineral oil (as kerosene) has been added.

This gives a stock solution, which must be diluted for use. The dilution can only be done with the aid of a hydrometer marked with degrees Baumé for liquids heavier than water, since the concentration varies according to the purity of the materials and the details of preparation. It is also customary in many places to buy the concentrated solution ready made, and the commercial preparations vary within even wider limits. The concentrated solution may give a reading of anywhere from 28° to 36° Baumé or even outside these figures, though from 32° to 36° appear to be most common for the commercial and 28° to 30° for the home-made. The strengths suitable for use vary with the plant to be sprayed and the season at which spraying is to be done, but they range for plants in leaf between about 0.5 and 2° Baumé, or a specific gravity of between 1.004 and 1.014. In England, a strength of 1.01 sp. gr. has been recommended for use on fruit trees, or, in cases where this strength has been found to cause injury, 1.005 may be used. The following table gives approximately the number of gallons of water that should be added to one gallon of a concentrated solution, between 28° and 36° Baumé, to make these solutions.

*Dilution table for concentrated lime-sulphur solutions.*

DENSITY OF THE CONCENTRATED SOLUTION IN DEGREES BAUMÉ.	NUMBER OF GALLONS OF WATER TO BE ADDED TO ONE GALLON OF THE CONCENTRATED SOLUTION.	
	To make 1.01 solution.	To make 1.005 solution.
28°	22	46
29°	23	48
30°	24	50
31°	26	52
32°	27	54
33°	28	57
34°	29	59
35°	30	61
36°	31	63

For use on dormant woody plants before the leaves appear, a strength of 1 gallon to 9 to 11 gallons of water gives satisfactory results.

*Self-boiled lime-sulphur.*

This is a less effective general fungicide than the last, but is recommended for spraying plants in leaf that are injured by either Bordeaux mixture or the concentrated lime-sulphur preparations. It is practically harmless even to delicate plants, and is very easily made.

Quicklime	..	..	..	8 lb.
Sulphur	..	..	..	8 lb.
Water	..	..	..	50 gallons.

Place the lime in a barrel and add enough water almost to cover it. As soon as slaking starts, add the sulphur. Stir continuously and add enough water to make a paste and prevent caking at the bottom. When the violent slaking is over, add more water so as to cool it, as prolonged heating is injurious. Then dilute to 50 gallons and strain, when it is ready for use. A sieve of 20 meshes to the inch is suitable for straining, and all the sulphur should be worked through, keeping back only the unbroken lime lumps. A short period of intense heating gives the best mixture, so large lots should be made up at a time, as a small quantity of lime will not produce enough heat.

In making up the lime-sulphur solutions, use only the best materials, freshly burnt stone-lime, and high quality, finely divided sulphur, which should not be allowed to get lumpy. The self-boiled mixture should be vigorously agitated while spraying. It is a weak fungicide and only valuable for spraying very delicate plants, such as peaches in leaf. Lime sulphur leaves an unsightly whitish deposit on the leaves, and is unsuitable on this account for ornamental plants. It is very adhesive, standing rain well. Injury may be caused by the concentrated solutions if used too strong, the leaves being scorched or caused to fall. In such cases very dilute solutions are required, or the self-boiled mixture may be tried. The ordinary sprayers used for Bordeaux mixture are partly composed of copper and cannot be employed for lime-sulphur sprays; brass alloy or steel (lead-lined with a lining of bitumen mastic) is preferable and can be used for Bordeaux mixture also.

**Powders.** **SULPHUR.**—This is used in the finely divided form known as "flowers of sulphur," chiefly against the powdery mildews (*Erysiphaceæ*). It is convenient for garden use, but is likely to be replaced in field practice by the lime-sulphur combinations. It is best dusted on by means of special bellows and, if possible, early in the morning while the leaves are still damp from dew, as it adheres better

than if the leaves are dry. The best results are got on fine, warm days, while rain is injurious, as the powder is readily washed off. Lime or soot is sometimes added, chiefly with a view to diluting the sulphur so that it will go further.

Various other substances and combinations have been prepared in powdered form for use as fungicides, but their use has not become general and it seems improbable that they will replace liquid sprays.

---





**PART II.**  
**SPECIAL DISEASES.**



## CHAPTER VI.

### CEREALS.

#### WHEAT (*Triticum* spp.)

**Rust** (*Puccinia graminis* Pers., *Puccinia glumarum* Erika. and Henn., *Puccinia triticina* Erika.).—Three different forms of rust attack wheat in India : black rust caused by *Puccinia graminis* ; yellow rust, caused by *P. glumarum* ; and orange rust, caused by *P. triticina*. In their total effects these parasites cause more damage than do any other crop diseases in the country.

Black and orange rusts are practically of world-wide distribution ; yellow rust is more restricted, being absent, for instance, from South Africa and Australia.

**BLACK RUST.**—*Puccinia graminis* usually does not appear on the wheat crop until late in the season. It is often not seen until March in northern India, a time when the wheat is in ear. Its onset is first marked by an eruption of elongated, brown pustules on the stalk, leaf sheaths, and leaves, the stalk being often most attacked. These pustules (uredo sori) may be a quarter of an inch or more in length and frequently run into one another. They very soon burst, exposing a brown powder consisting of multitudes of uredospores. Later on, teleuto sori develop. The teleutospores are often, indeed, produced in the same sorus as the uredospores, and as they are darker in colour than the latter, one can see the pustules gradually change from brown to black as the season advances. The position of the teleuto sori is similar to that of the uredo, and they burst through the epidermis in the same manner, exposing a black bed of teleutospores.

Each sorus is formed on a limited mycelium, the hyphae of which are about 3  $\mu$  in diameter and extend between the cells of the plant, sending small, round or branched suckers (haustoria) into them and feeding on them. Soon a mass of hyphae collects under the epidermis and, if early in the season, develops into a uredo sorus. From the base of the sorus, sometimes short, stout stalks arise, and a uredospore is produced at the end of each. The growth in size of these spores ruptures the epidermis and they are set free into the air. Each uredospore is an oval, brown body, measuring 25 to 30 by 17 to 20  $\mu$ , and consisting of a single cell with a thick wall, provided with tiny spines for anchoring the spore to the surface on which it falls. The wall is thinned in places to

form what are known as germ-pores, through which the germ-tubes emerge. The number of these pores in the present species is usually 4, arranged in an equatorial band.



FIG. 80. 1, Black rust of wheat (*Puccinia graminis*). 2-5, Yellow rust of wheat (*Puccinia glumarum*).

The uredospores germinate rapidly in water or moist air, sending out a germ-tube capable of developing into a new parasitic mycelium within the plant. A second germ-tube is often formed, but soon ceases growth. The germ-tube does not enter directly, but the tip swells up into an elongated appressorium, which measures about 27 by 9  $\mu$  and lies with its long axis covering the slit-like mouth of a stoma. From this, a narrow branch passes into the stoma and immediately swells up to form a similarly shaped "sub-stomatal vesicle." All the contents of the germ-tube collect in the latter, and soon a hypha arises from one end and grows towards a neighbouring cell, into which it sends a haustorium. Infection is now complete and the mycelium can continue its growth within the plant. From this mycelium, new pustules are formed in 10 or 15 days from the infection, and a fresh crop of uredospores is thrown off into the air. Since a single pustule may contain hundreds of spores, each capable in suitable weather of causing a new infection and new pustules, it is easy to understand how the fungus can multiply and spread through a field, until every plant is infected and even the ground is coloured by the shed spores.



Fig. 51. *Puccinia graminis*. 1, medial stage; 2, part of uredo sorus; 3, germination of uredospore; 4, part of telueto sorus; 5, germination of telueto spore. (2 and 6 after de Bary, 3 after Duggar, 3-5 after Eriksson and Hennings).

The telueto sori arise on the same mycelium, later in the year. The spores differ from the last in being firmly fixed on their stalks, and composed of two cells with a thick, smooth wall. They are chestnut-brown in colour and measure 40 to 60 by 15 to 20  $\mu$ . Each cell has a germ-pore, that of the upper being at the apex and that of the lower at

the side, just below the septum. The apex is rounded or pointed, rarely flattened, and its wall much thickened (up to  $12\mu$ ).

Unlike the uredo, these spores are not capable of germination immediately they are formed, but must go through a period of rest of several months. Hence they are not intended for rapid spread, but are "resting spores" to carry the parasite over to the following season. This they are known to do in other countries, but they seem to have lost their power of germination in the plains of India.

On germination, the teleutospore gives rise, from each cell, to a peculiar type of germ-tube called a promycelium. This is a short hypha, divided into four segments, from each of which a little branch (a "sterigma") grows out and bears at the end a small roundish sporidium. The sporidia fall off easily and are blown about. If they fall in a moist place, they germinate. The germ-tube which comes from a sporidium is unable to infect the wheat plant but can enter the leaf of the barberry, a common shrub in the Himalaya and other Indian hills. Entry takes place directly through the epidermis, not into a stoma. Inside, a mycelium is developed which causes a small yellowish patch on which two further types of fructification are produced, spermatogonia on the upper followed by aecidia on the lower surface.

The spermatogonia are little flask-shaped spore cases, which contain minute spores ("spermata") that appear to have no present function. The aecidia are yellow, cup-shaped receptacles, formed beneath the epidermis. Each is composed of a sterile, membranous wall ("peridium") enclosing chains of yellow aecidiospores arising from the base of the cup. With their growth, the epidermis is ruptured and the membranous envelope protrudes. This in turn bursts at the apex and is turned back, the torn edge forming a serrated rim to the cup. The spores thus set free are readily disseminated by air currents. They are roundish or angular, measuring 14 to  $26\mu$  in diameter, and have a spiny wall with about 6 germ-pores. They are capable of immediate germination but, like the sporidia, their germ-tubes cannot infect the plant on which they were borne. Their function is to carry the fungus back to wheat, which they can penetrate through the stomata to form a parasitic mycelium and ultimately uredospore pustules.

*Puccinia graminis* is a "heteroecious" rust, requiring two host plants, barberry and wheat, to go through its whole development, just as the malarial parasite requires in turn to infect a mosquito and man in order to complete its full life.

In some countries, the aecidial stage of the rust on barberry has been altogether lost, and probably in all, the fungus is able frequently to dispense with this stage. Thus in Australia, where black rust is common and has been known since 1825, its aecidial stage has never been found. South Africa and Ecuador are similar cases. In India, black rust occurs every year in the centre of the country, hundreds of miles away from the nearest barberries, as these are only found in the Himalaya and a few other of the higher ranges. The aecidial stage of *P. graminis* has been found on barberries near Simla and probably also occurs in the Kumaon Hills, but these localities are too far away from Central India to have any connection with the black rust of the wheat fields there. Besides, this parasite is one which is specialised on its host plants to a remarkable degree. It is known to occur on many cereals and grasses,

but has become split up into biological species (p. 61), so that the form on one host is not able to infect certain of the others. Black rust, for instance, attacks oats in many countries but the form on wheat cannot live on oats, nor that on oats on wheat.\* Hence it is common to find oat fields quite free from rust alongside wheat fields badly attacked by black rust. In fact, though black rust is so common on wheat and barley, it has never been found on oats in this country, as apparently the race which can live on oats has not yet been introduced, or at least has failed to become acclimatised. Now the æcidium found on barberries near Simla has been shown to belong to the black rust of a jungle grass and is not connected with the black rust of wheat. Similarly, the barberry æcidiospores in Kumaon could not be got to infect wheat and probably belong to some other race of the parasite. Hence, in India, the æcidial stage is of no account to the parasite but the latter passes all its life on wheat only. It is probably in connection with this fact, that the teleutospores cannot be germinated in India or South Africa; having lost the æcidial stage, their germination would serve no useful purpose.

**YELLOW RUST.**—*Puccinia glumarum* is usually of earlier appearance than *P. graminis*, coming out as a rule before the grain is formed. In mild attacks, the uredo pustules are found chiefly on the leaves, but in more severe cases they appear on the sheaths, stalk, and glumes as well. The green colour of the leaf fades in long streaks, on which rows of small uredo pustules appear. Each row consists of a series of oval, lemon-yellow pustules arranged end to end and each distinct from that above and below. In severe attacks, this serial arrangement tends to be lost, large patches being covered with crowded pustules. The uredospores do not break through the epidermis as quickly as in the other rusts, but do so eventually and quantities of yellow spores are shed.

The teleuto sori appear later as dull black patches or spots, chiefly on the under surface of the leaves, but also on the other parts. Like the uredo, they are often arranged in rows. They do not burst through the epidermis as in black rust, but remain as flat black crusts.

\* In the United States, it has been found that though the form on wheat cannot infect oats directly, it can do so if first passed through barley. In India, barley is sometimes attacked by the fungus from wheat, and the absence of the rust on oats near rusted barley fields indicates that even in this case it cannot infect oats here. Climate may have something to do with this difference between the rust in the two countries, for it has been observed in South Africa that barley is highly susceptible to the attack of black rust from wheat in summer, while in winter it is immune. Indian oats grown in the Transvaal were badly attacked by *P. graminis*, which presumably came from the local oat crop.

The uredospores are nearly round, not oval as in black rust. They measure 23 to 35 by 20 to 35 $\mu$ , and have a colourless wall provided with fine spines and with 6 to 10 scattered germ-pores.

On germination, a small, fragile appressorium forms over a stoma. The tube of entry swells up to a large, thick-walled, cylindrical sub-stomatal vesicle, up to 19 $\mu$  broad and placed just below the slit. From this an infection hypha aneas. The mycelium extends rapidly by means of longitudinal "runners," which have no haustoria and may remain unbranched for a considerable distance. Other short, branching hyphae, with club-shaped or arborescent haustoria, obtain food from the cells. The hyphae are very thick, 10 to 19 $\mu$  across, until they collect beneath the stomata to form sori, when they are only 3.5 to 6 $\mu$  in diameter.

The teleutospores are dark brown, often flattened at the top, and measure 35 to 63 by 12 to 20 $\mu$ . They may occupy all the sorus or may be broken up into groups by rows of sterile cells or paraphyses.

The further development of this rust has probably been lost. The teleutospores germinate after a rest and produce promycelia with

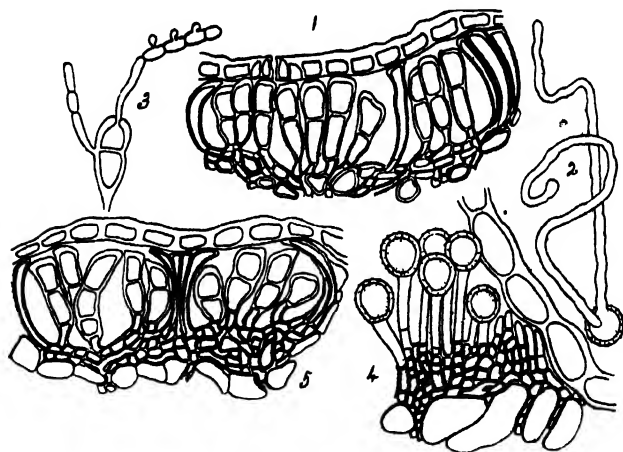


FIG. 52. *Puccinia glumarum*: 1, section through a teleuto sorus,  $\times 320$ ; 2, germination of uredospore,  $\times 320$ ; 3, germination of teleutospore,  $\times 320$ . *Puccinia triticina*: 4, section through a urredo sorus,  $\times 320$ ; 5, section through a teleuto sorus showing 1 to 3-celled spores,  $\times 320$ . (1-3 after de Jacewski).

sporidia, but these sporidia cannot infect any plant, so far as is known. It is probable that the fungus was originally heteroecious, but that the scidial stage, becoming of little value, has been lost.

**ORANGE RUST.**—*Puccinia triticina* is the earliest rust to appear in Bengal, the United Provinces, and probably elsewhere. It has been



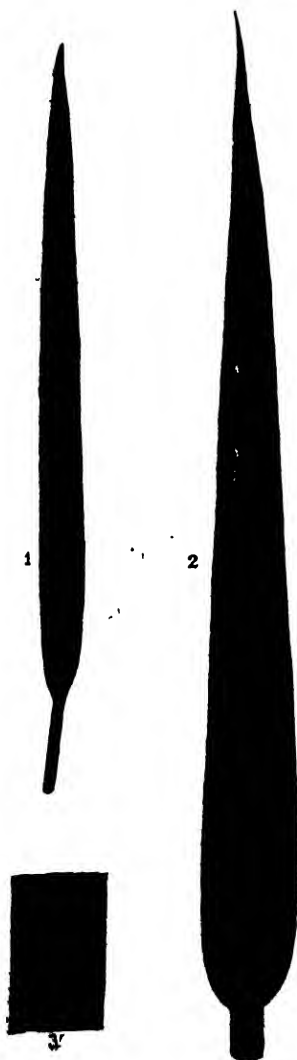


FIG. 53. Orange rust of wheat (*Puccinia triticina*): 1-2, uredo stage; 3, teleuto

noticed in Pusa as early as the last week of November when the crop was between five and six weeks old, but this was an exceptional case and it is rarely seen before the beginning of the New-year. The uredo pustules come out on the leaves as a rule, being scarce on the sheaths and stalks. They burst out on the upper surface as points of a bright orange colour. They are never in rows like those of yellow rust, but are grouped in small clusters or irregularly scattered. When old, they are not easy to distinguish by colour from yellow rust, but can generally be known by their irregular arrangement. The sori burst early and shed their uredospores.

As a rule, teleutospores are not formed. Some years they may be searched for with the greatest care unsuccessfully. In 1904, however, they were quite common and have appeared on several other occasions. This would seem to indicate that their formation depends on some special climatic conditions. They resemble those of yellow rust, the chief difference being that they are not arranged in rows.

The uredospores are similar in size and shape to those of *P. glumarum*, but have a brownish wall. Infection occurs as in the other forms, but the sub-stomatal vesicle is round or elliptical and the hyphae resemble those of *P. graminis* rather than *P. glumarum*.

The teleuto sori are broken up into more numerous compartments by partitions of paraphyses, but otherwise resemble, as do the spores, those of yellow rust.

As with the last species, no further stage than the production of sporidia is known; the sporidia are unable to infect wheat, and the second host plant, if one ever existed, appears to have been lost.

*Table of the chief naked-eye characters by which the three rusts can be distinguished.*

Black rust. ( <i>P. graminis</i> .)	Yellow rust. ( <i>P. glumarum</i> .)	Orange rust. ( <i>P. triticea</i> .)
Attacks the stalk most severely, then the leaf sheaths, leaves, and ears.	Attacks the leaves most severely, then the leaf sheaths, stalks, and ears.	Attacks the leaves almost exclusively, rarely the leaf sheaths, very rarely the stalks.
Uredo pustules large, elongated, running together and bursting early, throwing up large fragments of the epidermis in doing so. Colour warm brown, becoming darker gradually as teleutospores are formed in the same pustules. Found on all green parts of the plant.	Uredo pustules small, oval, do not usually run together, burst late and with little displacement of the epidermis. Almost always arranged in long rows. Colour lemon-yellow. Found on all green parts of the plant.	Uredo pustules small but often larger than in yellow rust, oval or round, do not usually run together, burst early with a fringe of broken epidermis around them. Never arranged in long rows. Colour bright orange when fresh. Found chiefly on upper surface of leaves.
Teleuto pustules like uredo but black. Burst rather early. Found on all green parts of the plant, but least on the leaf blades.	Teleuto pustules like uredo but more flattened, dull black, and often run together. Do not burst through the epidermis. Arranged in rows. Found chiefly on under surface of leaves but also on all green parts of the plant.	Teleuto pustules often absent, when present resemble uredo but more flattened and dull black. Do not burst through the epidermis. Found chiefly on under surface of leaves, very rare elsewhere.

The greater part of the thirty million acres or so under wheat in India is found north of a line joining Bombay and Calcutta. In the whole of this area it is grown as a cold weather crop, being sown in October-November and harvested sometimes as early as February in the southern parts and as late as May in the Punjab and the Kumaon hills. In the extreme north and north-west there is little growth in the cold weather, and in Kashmir and Baluchistan the crop is reaped in July. During the greater part of the hot weather and all the monsoon months, no wheat is to be found in the plains. Hence the parasite must have some method of living over in the interval between the harvest of one crop and the period when rust first appears in the next crop. Black

rust may do this in the north-west Himalaya by passing on to barberry bushes through its "resting" teleutospores, and there producing the aecidial stage which can carry it back to the wheat. But even in that area, it is doubtful if the barberry carries the race of the parasite which is found on wheat, while in the plains there is no plant on which the aecidium can live; the aecidial stage has been lost, as in Australia, and the fungus is only found in the uredo-teleuto stages. In the case of yellow and orange rusts there is no aecidial stage, so that here also there is no possibility of the parasite persisting by means of its teleutospores. How then do these fungi tide over the unfavourable season when they have no wheat to feed on? No satisfactory answer can as yet be given to this question, though various explanations have been attempted which may serve to illustrate some of the difficulties frequently met with in accounting for the attack of annual parasites.

One view is that the parasite can live on accidental plants of wheat which come up naturally in the hot weather and rains. These are, however, very rare, probably quite absent in the hotter parts of the country. Attempts to grow wheat continuously all the year round at the Cawnpore Experiment Station proved very troublesome and no rust appeared on any plant after July up to December. At Poona, on the other hand, wheat was successfully grown throughout the summer months, the climate being comparatively temperate. The first sowing was inoculated with black rust in May and bore uredo pustules in a little over a month. Further sowings became spontaneously infected up to December. In Europe, North America, South Africa, and Australia, also, the uredo stage has been found throughout the year; and a violent outbreak of rust on winter wheat was even recorded in December, 1913, in Lorraine. Experiments have, however, shown that heat is more destructive than cold; five minutes moist heat at 113°F. or a few hours exposure to the sun when the shade temperature is 95°F., is enough to kill all the uredospores. Attempts to preserve the spores for a few months in the early part of the hot weather have been unsuccessful. Hence, even if stray wheat plants do occasionally live through the summer months in the cooler places, it is very unlikely that they do so in the Indo-Gangetic Plain or that they could possibly become rusted if they did.

Another view is that it can live on some other grass during these months. Black and yellow rusts have many grass hosts. Repeated search has failed to disclose any suitable host in the plains and even if one exist, the parasite is unlikely to survive the heat. *P. glumarum* has been

found on *Phalaris minor* in the Punjab and United Provinces and on *Brachypodium sylvaticum* in the Himalaya. The former is a cold weather grass and is absent from much of the wheat area. *P. graminis* has been found also on *Brachypodium sylvaticum* and on *Festuca gigantea* and *F. kashmiriana* in Kashmir. These grasses do not extend into the plains and can therefore play no part in carrying rust in the main wheatgrowing tracts.

A third view is that the uredospores may live in the soil until the new crop is ready for infection. This is possible, but no direct evidence has been obtained in its favour. If it occurs, it is hard to understand why black rust does not appear soon after the young wheat is up in October, instead of waiting until February or March. As already said, attempts to keep the uredospores alive in the hot weather have failed; and it is known that the teleutospores cannot infect wheat. The "uredospore theory" of the origin of wheat rust each year has been gaining ground of late, however, and undoubtedly serves to explain the circumstances in some countries, as the United States, where wheat is found all the year round in one part or another of the area. Even in India, it is far from having been disproved, as we know too little of the conditions which affect the vitality and power of germination of these spores. Thus in cold climates, uredospores of *P. graminis* have been kept, under snow, capable of germinating in part, from December up to March. *P. triticea* uredospores have also been germinated in the March following their formation, in the United States, and after 2½ months in Europe. In certain cases too, it is known that the incubation period between the time of infection and the appearance of new pustules may be greatly lengthened. In Poona, as noted above, inoculations in May gave sori only after 35 days. In Austria, wheat infected in October-November bore pustules first at the end of March, when exposed to the winter cold. In such cases, there is evidence that the mycelium, after infection, passes into a dormant condition and only resumes growth at a later stage, and it is clear that wherever wheat occurs all the year round this may be a valuable aid in carrying the fungus through the bad seasons. It cannot be a factor of importance in India.

A few mycologists believe that the parasite infects the grain and lives in symbiosis with the protoplasm of the grain cells; when these are sown and germinate, it is supposed to grow, still mingled with the host protoplasm, and to break out as a mycelium, bearing uredo pustules, only when the conditions become favourable for a parasitic life. In the

potato disease and some cereal smuts the mycelium undoubtedly lives from one generation to another inside the tuber or the grain ; but it can be found on microscopic examination, whereas in the grain rusts no mycelium is seen until shortly before the pustules appear. Hence it is necessary to believe that the parasite is so mingled with the host cells as to be invisible except under very special treatment. Such evidence as there is in favour of this " mycoplasma " theory (which would fully explain all the facts observed in India) has been vigorously assailed. On the technical side, the work has been criticised by more than one competent observer ; on the experimental, several workers have found that it is possible to grow highly susceptible wheat entirely free from rust, provided it is enclosed in spore-proof cages. At Cawnpore, for instance, a large number of plants (some 190) were raised in this manner and only two minute pustules appeared in two cages, which were found to have been imperfectly closed. The most recent exponents of the theory have based their conclusions in part on work carried out with the hollyhock rust (*P. Malvacearum*) and it has been claimed that sori can be induced to form in the absence of external infection, provided the amount of free water in the tissues is reduced. The failure in previous experiments is ascribed to the humidity having been kept at too high a level.

Though no mycelium can be detected in the green parts until just prior to the eruption of uredo sori, it has recently been found that a certain proportion of the grains of wheat, and still more of barley, bear sori of *P. graminis* and *P. glumarum*, more or less completely buried in the grain-coats and facing inwards as a rule. On germination, spores from these sori may be carried up on the young shoot and, if still living, might start an attack of rust. Further, a mycelium agreeing with that of the rust has been found in the primary shoot and roots from such grains, but new sori have not yet been observed to develop in the young seedling and until this is done no special weight can be attached to these observations.

In Bengal, Bihar, and the United Provinces, all three rusts usually attack the wheat every year. Early in the season, orange rust may alone be found ; a little later, it is combined with yellow rust ; later still, all three are often seen on the same plant. In the Central Provinces and Bombay, it is doubtful whether orange rust occurs in ordinary years, but in years of bad rust it extends well into the former area. In the Punjab also, it appears to be less common than the others. Owing to the different kinds occurring so frequently together in bad years, it is difficult to find

out which does the greatest damage. In Bengal, it is probable that *P. tritici* is usually the worst; in the Central Provinces, perhaps *P. graminis* or *P. glumarum*. A few experiments made some years ago seem to show that black rust is not as harmful to the grain as the others. Thus it was found that ten healthy grains weighed as much as thirty from plants attacked severely with black rust and as sixty attacked with one of the others (whether yellow or orange is not certain). In Australia, on the other hand, everyone seems agreed that black rust does much more damage than orange.

Though rust is found every year in practically every wheat field, the intensity of the attack varies greatly. Whether a generalised epidemic breaks out or not, depends to a great extent (as shown above on p. 129) on the weather conditions of the year.

Localised attacks depend for their severity on a number of somewhat complex factors. Some are external, such as the humidity of the air within the crop itself. This is probably the reason why severe rust frequently follows over-irrigation during the growing period. In certain of the less well-drained soils, heavy irrigation is by itself sometimes enough to induce rusting. In other soils, such as those of the Punjab Canal Colonies, no ordinary irrigation produces water-logging and the supply of water does not seem to influence rust to such a marked degree. In Pusa, it has often been observed that black and yellow rusts are copious in the denser parts of the crop and less in the light crop near trees at the margin of the fields. Direct measurements showed that both the soil moisture and the humidity of the air within the crop (above ground) were higher in the heavy crop, the latter being probably the controlling factor. Another factor concerned in these cases is possibly the turgidity of the cells, due to their high moisture content; this, though an "internal" factor, is dependent on external conditions such as the supply of water to the roots and the freedom of transpiration. These factors appear to act by promoting the germination of the uredospores and facilitating the entry of their germ-tubes. More obscure are the factors which determine the resistance of the plant itself to attack. The effect of certain manures has already been discussed (p. 122) as also the inherent partial immunity of some varieties (pp. 118, 126). In some cases, early outbreaks of rust have been observed where the crop has been sown on too dry a seed bed; the only attack before Christmas hitherto seen at Pusa was under such conditions. So also the high temperature associated with strong west winds may bring on a severe attack of black rust in late

wheats. The rust-resistant Einkorn variety became, under such conditions, as rusty at Pusa as any ordinary susceptible Indian wheat.

The amount of injury caused by an attack depends on another set of factors, which must not be confused with the above. Weak-stawed wheats suffer more from stem attacks of black rust than those with strong stalks, though the *degree* of attack may be the same. The rapidity of the attack at certain periods is also important, the plant resisting better when the onset is slow. Wheat in the Punjab that has become rusted as a result of weather conditions in the cold months has been observed to be unable to stand the strain caused by the rapid onset of hot weather at the end of March. The grain shrivels as a result of too rapid ripening, whereas similarly rusted wheat can give a good crop if it ripens slowly. Hot, dry spells are said to increase the damage in South Africa, and drought following heavy late spring rains in Australia.

Direct treatment of the rusts is not satisfactory. The most promising method of checking the injury is by the introduction of rust-resisting varieties of wheat into cultivation. It has been shown that resistance to rust is, at least in the case of yellow rust, a "Mendelian" character and hence that hybridisation with a view to producing resistant varieties is likely to lead to results of great value. The objects to be aimed at are to produce varieties which combine resistance to the most important local rusts with other desirable characters, such as heavy yield, good quality of grain and so on. Such wheats have been already developed in various countries and had indeed been raised (as in Australia) before the laws governing their production were understood. In India also, the work has been taken up by the Agricultural Department. It is practically the only way by which we may hope to check the disease.

Black and yellow rusts also attack barley in India; the races on barley are probably unable, however, to infect wheat in either case, though black rust can do so in the United States. Orange rust is confined to wheat.

**Loose smut** (*Ustilago Tritici* (Pers.) Jens.).—The loose smut of wheat, so called to distinguish it from the "stinking" smut or bunt described below, is a fairly common disease in most of the wheat-growing provinces, without often becoming epidemic or doing much damage. Its distribution throughout the world practically corresponds to that of its host plant.

Infected plants cannot be recognised until they come into ear, which they do just before the rest of the crop. It is then found that in place

of the normal ear, a blackish, powdery mass of spores appears. These spores develop in the young spikelets, covered by a delicate silvery

membrane which bursts usually before the head emerges from the sheath, forming very dark olive-brown, powdery masses in place of the spikelets, of which only the ends of the awns ordinarily escape transformation. The spores separate easily from the plant, and in dry weather may be almost all blown off, leaving a bare stalk (the rachis) behind.

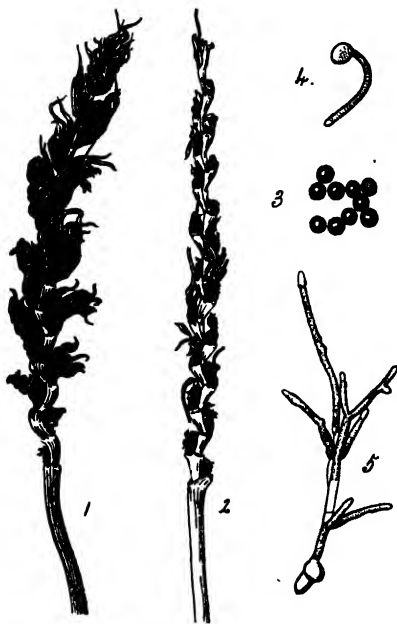


FIG. 54. Wheat smut (*Ustilago Tritici*): 1, fully smutted ear, nat. size; 2, later stage of same; 3, spores,  $\times 500$ ; 4-5, germination of spores,  $\times 500$ . (3-5, after McAlpine).

The spores are pale olive-brown, lighter on one side, spherical or occasionally oval, and measure 5 to 9  $\mu$  in greatest diameter. The wall has fine spines, especially on the lighter side. Germination occurs in moisture by a germ-tube, which soon dies unless the spore has fallen on the feathery style of a normal wheat flower. Under no other known circumstance can the fungus escape premature death. In a crowded wheat field the chances are not so adverse as might be thought. In dry weather clouds of spores

are thrown into the air. In dry weather also the glumes of sound flowers are rather widely open and the styles exposed to the spore dust. In wet, cold weather, however, both spore diffusion and the opening of the glumes are diminished and few spores reach their destination.

The spores germinating on the style send their germ-tubes into the latter only when its ephemeral cells have begun to collapse and dry up. The germ-tubes then enter in and between the cells and pass down to the lower, still living, part of the style. Here many are checked but others continue through intercellular spaces and the channels left by pollen tubes, until in a week or so the cavity of the ovary is reached. The ovules are next penetrated through their integuments (the outer of which has by now partly disintegrated), entry taking place through small intercellular spaces. The integuments ordinarily become cutinised and impermeable about 10 days after the normal time of infection, so that successful penetration of the ovule usually occurs between the 7th



and 10th day. The infection tube then passes into the space between the endosperm and the nucellus, where it branches freely for the first time. In about 3 weeks, branches have reached the lower end of the raphe, and they pass round the bottom of the endosperm to reach the scutellum and penetrate the embryo. Here some of them grow along the rudimentary vascular bundles to occupy the part between the apex and the root, it being now about 4 weeks after infection. A month later all parts of the embryo, except the root, contain hyphæ and there is a very copious mycelium in the scutellum. The hyphæ are mostly only 2.5 to 3  $\mu$  in diameter, except in the scutellum where they are often somewhat swollen; growth is exclusively intercellular; there are no haustoria; and the host cells are not affected in the slightest degree by the presence of the parasite. In the ripe grain, the hyphæ are thick-walled, oily, and irregularly swollen; though no haustoria are found, the cell walls are indented in places.

The parasite lies thus dormant within the grain until the latter germinates, when it grows just behind the growing point and keeps pace with the apex of the plant. As the ear forms there is a great accumulation of hyphæ, which give rise to the spore-masses above described.

Occasionally spore formation occurs on the leaf blade or even on the stem. This has been observed in Egypt and Australia, in the latter only on Egyptian wheat. It has also been found at Cawnpore and Surat on local wheats.

The spores retain their vitality for 5 or 6 months, a much shorter period than in the other cereal smuts. They can germinate freely in water or nutrient solutions, giving a small, branched mycelium.<sup>77</sup> No conidia are formed on this and the fungus does not seem to be capable, like most of its allies, of a saprophytic life in the soil. In nature, immediate infection alone is likely to succeed, and prolonged vitality or a period of saprophytic life would be of no advantage to the parasite.

The most satisfactory treatment at present known is to soak the grain in water hot enough to kill the internal mycelium without seriously injuring the seed. As it is very troublesome except with small lots, it is recommended to use it for the treatment only of a seed-plot, the produce of which is to be kept for sowing the next season's field crop.

The seed-plot should be large enough to allow for loss in cleaning and selecting the seed and still provide enough for the field sowings. It should be located so that the prevailing winds at flowering time will not blow smut spores into it, and preferably not nearer other wheat than 500 yards; wind breaks (trees) or large fields of other crops around it will diminish the danger of external infection.

The seed for the seed-plot should be first soaked for 4 to 6 hours in water at from 68° to 86°F. (the ordinary room temperature of many parts of India at sowing time), then placed in small sacks or baskets and immersed in water at 129°F. for 10 minutes. It is very important that all the grain should be rapidly and uniformly brought to the required

temperature and kept as near as possible at this point for 10 minutes. This is best done by having two tubs of water, one heated to about 120°F. into which the grain is plunged for a couple of minutes so as to warm it, and the other at exactly 129°F. in which it is then placed for 10 minutes. A thermometer should be kept in the second tub and hot water added as the temperature falls, the contents being kept stirred meanwhile. A temperature below 124°F. is ineffective, even if a longer treatment is given, and above 130°F. or more than 10 minutes at 129°F. injures the seed. It should then be well dried and sown soon to avoid mouldiness or deterioration. Since even good sound seed has from 6 to 10 per cent. of the grains injured by the heat, it is necessary to increase the sowing rate. Bad seed may suffer more heavily.

The treatment is usually satisfactory, though somewhat irregular. It should be carried out whenever the number of smutted ears much exceeds 1 per cent. In northern India it is usually below this figure, but in the Central Provinces nearly 10 per cent. has been reported. The disease is probably on the increase, since no measures are taken to check it, and treatment may require to be more considered in the future.

In the absence of special treatment, it is naturally of advantage to sow grain only from smut-free fields where possible. Seed treatment with surface disinfectants, such as are used for allied fungi, is of no value, since the fungus is already within the grain at the time of sowing.

The fungus is only known on wheat.

**Bunt** (*Tilletia Triticæ* (Bjerk.) Wint. and *T. levis* Kuehn).—This is usually a much more serious disease of wheat in the countries in which it occurs, than the loose smut. In India, it is confined to the north-west, being extremely abundant in Kashmir and the western Himalaya, common also in parts of Baluchistan, and extending into the Punjab. In the east, centre, and south of the country it is unknown, being apparently unable to stand the heat at sowing time in these areas.

Affected plants ripen usually more rapidly than sound plants and the ears are of a darker green colour and more open. Except for these signs, not always easy to detect, there is little to distinguish bunted from normal wheat. At full ripeness the grain is dark and the dark colour is visible between the glumes, which are more spread out than usual. The ears themselves remain erect and rigid, instead of bending over like the sound ones.

If the grain be examined more closely, it is found that the inside is filled with a greasy spore-mass, smelling strongly of rotten fish (due to the chemical compound, trimethylamin) and entirely replacing the grain contents while leaving the grain coats intact. Unlike loose smut, the spore-mass does not dry into a powder capable of being blown about by the wind; it becomes hard and may remain unbroken through the processes of reaping, threshing, and winnowing. Usually, however, the spore-balls are ruptured at some period during the handling of the grain, generally at threshing, and the spores then come into contact with sound grains, to the outside of which they adhere. Every place which has held bunted grain, and every article which has touched it, becomes contaminated and can infect clean grain. Naturally the soil of the wheat field also becomes infected during reaping.

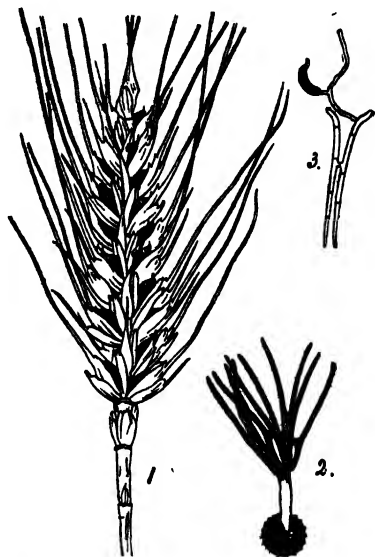


FIG. 55. Bunt (*Tilletia Tritici*): 1, bunted wheat ear, nat. size; 2, germinated spore with paired sporidia; 3, paired sporidia germinating with formation of secondary sporidium. (2 after Tulasne, 3 after Brefeld).

The two species of bunt, *Tilletia Tritici* and *T. levis*, differ only in their spores, the former having a reticulate wall, the latter a smooth one. In *T. Tritici* the spores are generally round, measuring from 15 to 20 $\mu$  in diameter, while in *T. levis* they are more irregular, round, elliptical, or angular, and from 16 to 25 $\mu$  across. Both often occur on the same stool and even in the same ear and they agree in their life-history.

The spore germinates in water or moist air, producing a stout promycelium which grows up into the air. At its tip, a cluster of 4 to 12 long, narrow, curved sporidia is formed. Its length is variable according to the depth of the liquid, as the sporidia are only formed in the air. These usually become united in pairs by lateral branches and then germinate by a fine hypha, which in turn may bear a secondary, sickle-shaped sporidium at its end. Usually the saprophytic life is limited to this short growth and the germ-tubes die early if they do not encounter a young wheat plant. Sometimes, however, as in nutrient solutions, or in the presence of abundant organic matter such as cattle manure,

the saprophytic life may be prolonged; the germ-tube from the sporidium branches and produces a mycelium of very fine hyphæ; and on aerial branches from this, numerous secondary sporidia, each capable of infecting a wheat seedling, may be formed.

Infection can only occur when the germ-tube of a primary or secondary sporidium comes in contact with a very recently germinated wheat seedling. Entry is effected by the hypha growing through the epidermal cells of the primary shoot below the soil level, and susceptibility is limited to the period of about 8 or 10 days between the moment of germination and the emergence of the first green leaf from the colourless bud-sheath ("coleoptile"). This period varies somewhat according to the variety and also the temperature. It is very short, for instance, in the variety known as Early Ohio and at ordinary temperatures this kind is almost immune. At low temperatures (near freezing point) wheat will germinate, though slowly, but the bunt spores not at all. The rate of germination of wheat increases up to 77°F., when it is at its maximum. The spores germinate best at a moderate temperature (45° to 65° F.), but are always rather slow, so that above a certain point the wheat is often able to get over its susceptible stage in time. Hence infection is most frequent at moderately low temperatures, as some figures obtained in France by sowing at different dates will indicate. Infected grain sown on October 1st gave only 1 per cent. of bunt; on November 10th, 10 per cent.; on November 22nd, 80 per cent.; on February 10th, 30 per cent.; and on March 10th, 5 per cent. Even the Early Ohio variety has been found to have 62 per cent. bunted under certain temperature conditions.

Successful infection is also influenced by soil moisture at the time of sowing. The seed bed may be just damp enough to allow the spores to germinate while insufficient for the wheat. In this case the germ-tubes perish before infection can take place.

After infection of the young shoot a mycelium is formed, which, as in loose smut, does not seem to cause any injury to the cells. If hyphæ reach the neighbourhood of the growing point, they keep pace with the lengthening plant, being found chiefly in the loose cells of the pith. As the ears develop the mycelium accumulates in the young ovaries; the cells are crushed and replaced by masses of hyphæ and finally the whole becomes transformed into a ball of spores. It is believed to be not uncommon for the fungus to fail to keep pace with the growing plant, so that successful infection does not necessarily result in the formation of bunted ears.

It has been found that though the spores in unbroken smut-balls will retain their vitality for 8 years if kept dry, and isolated spores for at least 3 years, still in moist soils all will have germinated after a few months. Under ordinary conditions, therefore, they have lost their vitality by the time the next wheat crop is sown. When, however, the soil is rich in organic matter, such as farm-yard manure, the fungus is capable of prolonged life as a saprophyte, though even in this case it often dies soon, as neither the mycelium nor the sporidia can survive drought. Further it seems probable that great cold keeps the spores alive in the soil. They are known to withstand freezing, provided they have not germinated (frost kills the germ-tubes) and in the extreme cold of western Canada the succeeding crop is believed to be often attacked by spores left in the soil. In eastern Canada, as in Europe, the intermittent heat and cold seems to be effective in freeing the soil of the parasite, and soil infection is believed to be rare. This would apply to the greater part of the infected area in India.

In practice it may be taken that by far the greater proportion of cases results from bunt spores adhering to the grain and germinating when the latter is sown. A certain amount may also come from manure heaps, but it has been found that passage through animals destroys the germinating power of the great majority of the spores. This effect is less with pigs than with other farm animals, but the danger from this source in India would not be great.

There is some difference of opinion as to the effect on the health of animals and man of eating bunted grain or flour. Experiments in Germany with pigs, cows, horses, sheep, fowls, etc., failed to give any definite case of ill effects, but it was concluded that heavily smutted grain should not be given to pregnant animals or to those already subject to intestinal troubles. In Australia, cases have been recorded of heavy loss on poultry farms from the reduction of eggs when the fowls were fed on bunted grain, and in Europe, from time to time, farm animals are said to suffer from paralysis and inflammation of the mucous membranes from the same cause, and it is even claimed that the spores may reach the blood and lymph and ultimately cause death.

It is quite common to find only some ears of a stool affected and even in these ears some grains may be sound. This may be due to the internal mycelium failing to pass into the growing points of some of the new shoots, or having infected them, it may fail to produce spores.

The losses caused by bunt are due chiefly to the actual replacement of the grain contents by the fungus. There are also many cases of infection that do not progress to spore formation but that shorten the straw and lead to sterility of the shoot. Further, the presence of bunt in the grain lowers its trade value, in Australia it is said by as much as sixpence to a shilling per bushel when bad. The disease was estimated to have caused a loss of £50,000 on the year's crop in Victoria in 1898.

From what has been said above, it is evident that if the spores adhering to the grain coats at the time of sowing are prevented from germinating and infecting the young shoots, the succeeding crop should be free from bunt. This can be done by several substances, but only two, copper sulphate (bluestone) and formalin, are extensively used.

*Copper sulphate.* A solution of 1 lb. copper sulphate in 5 gallons water (equal to 2 per cent.) is prepared, and this is sprinkled over the seed grain spread out on a floor, the heap being repeatedly turned over until every grain is wetted. A few minutes' sprinkling will be enough,

if the seed is well turned over. It is then dried by being spread out and turned over a couple of times, and is ready for sowing. An alternative method is to immerse the seed completely in a barrel full of the bluestone solution until all the grains are wetted (about a minute), stir up well, and skim off all the bunt balls, which will float. Then dry quickly and sow. This is preferable to the first, especially when there are many unbroken bunt balls, for the treatment does not kill spores inside such balls and consequently, if they break afterwards, as they are liable to do, they will re-infect the seed. The bluestone solution must not be placed in a metal vessel, but in a wooden tub or barrel. It can be used over and over again if kept from evaporating. The treatment is known to reduce the germinating power to some extent, as the coating which remains on the grain injures the delicate primary shoot. The injury is slight if the grain is sown within 24 hours of treatment, but increases if it is kept for several days. Some recommend that the seed, after being dried from the bluestone bath, should be immersed for 3 minutes in a weak lime-wash made by slaking about 1 lb. freshly burnt quicklime in 20 gallons water. This neutralises the injurious action of the copper sulphate and effects a saving of about 6 per cent. in the seed rate. The same results can also be obtained by the methods in use in France and Germany described on p. 182 below, under oat smut.

*Formalin.* A solution is prepared of 1 lb. commercial formalin (which is a solution containing about 37 per cent. of formaldehyde) in 40 gallons water. With this, the seed is thoroughly wetted by sprinkling (40 gallons will do for about 80 maunds of grain) and turning over on the floor, until every grain is treated, and then the heap is covered with damp gunny bags for about 2 hours to prevent evaporation. At the end of this time it is spread out to dry and should be sown early. Or, as an alternative, the grain is entirely immersed in a vessel (which may be of any convenient material) for 10 minutes, being well stirred and having all the floating bunt balls and débris skimmed off. It is then taken out and kept moist for 2 hours as before. If there are many unbroken bunt balls, this last method is to be preferred.

Either of these two treatments may be used with equally good results. The formalin treatment is increasing in popularity in many places, owing to the convenience in handling it. It is also less liable to injure germination, especially if the treated grain be sown promptly. Considerable injury may be caused if there is a delay in sowing, but this effect is said to wear off again after some weeks. The safest rule is to sow within 24 hours.

Whichever is used, great care is necessary to avoid re-infection of the grain after treatment. Everything which has come into contact with bunted grain becomes contaminated by the shed spores and these spores may get back to the seed through carelessness. Bags used to store bunted grain should not be again used after treatment, unless they have been immersed in the formalin solution to kill any adhering spores. In the same way, the treated grain should not be put into a store that has had diseased grain in it, without first spraying or sprinkling the inside with the fungicide. The same applies to carts. Even with these precautions some bunt balls may reach the grain, and if broken during sowing may cause infection. Hence it is wise to avoid using severely bunted grain for seed. It is probable that in a locality like the Kashmir Valley where, in 1908, bunt was observed to be very common, a single year's treatment would not entirely get rid of the disease. It would be necessary to carry it out for several years before the danger of re-infection became slight.

Should bunt and loose smut occur together, the hot water treatment described under the latter will give a seed crop free from both diseases.

Wheat varieties differ to a certain extent in their liability to bunt. It is not yet clear how far the partial immunity of some sorts is due to a direct power of the cells to resist attack, or to such indirect causes as the rapid germination of the Ohio wheat mentioned above. The breeding of bunt-resisting wheats is being attempted with some success in certain places, as in Australia, and may become an important line of attack in the future.

**Flag smut (*Urocystis Tritici* Koern.).**—This disease is confined to the Punjab in India, while outside India it is only known in east Asia and Australia; it is therefore of much more restricted distribution than the other smuts of wheat. Up to date, it has not been reported as causing much damage in this country, but the experience in Australia, where in some districts it is not rare for half the crop to be lost and a loss of 10 per cent. is common, shows that its importance should not be under-estimated.

As the name indicates it is usually found on the leaf blades; it is also common on the sheaths and occurs sometimes on the stem and very rarely on the ears. As a rule, every shoot of the plant is infected; their leaves become twisted up and assume a drooping habit, which is followed by withering; soon they are cast off and often the whole plant early dies down. Very frequently the entire stool bears no grain, the ear being replaced by a twisted mass of leaves. When grain is formed, it is usually

much shrivelled and valueless. The parasite first shows outwardly in long, grey, slightly swollen bands, parallel to the veins of the leaf. These then rupture and expose a black, powdery mass of spores.

The spores differ considerably from those of loose smut or bunt, being formed in tiny balls consisting of from 1 to 4 fertile bright brown spores, surrounding which is a layer of light-coloured sterile cells. The spores are spherical or oval and 9 to 16 $\mu$  in diameter, the spore-balls being up to 40 $\mu$  across. The sterile cells are smaller and more elliptical.



FIG. 56. Flag smut of wheat (*Urocystis Tritici*). 1, affected shoot,  $\times \frac{1}{2}$ ; 2, part of affected leaf, nat. size; 3, spore balls,  $\times 330$ ; 4, spore ball germinating,  $\times 330$ . (3-4 after McAlpine).

Germination occurs by the protrusion of a promycelium, or short 1 to 3-celled germ-tube, which bears a cluster of 2 to 6 sporidia at the apex. The sporidia are at first cylindrical and unseptate, then often become 1 or 2-septate. They measure 12 to 15 by 3 $\mu$  and germinate without falling off by a slender germ-tube, which may be of considerable length, and represents the infection hypha. Secondary sporidia are not formed.

Infection occurs below ground as in bunt. Experiments indicate that not only may the primary shoot be infected but also, at a later date, the lateral buds below ground from which the secondary shoots of the stool arise. The spore-balls, with their envelope of sterile cells, are well adapted to retain their powers of germination for a long time. It has been found, for instance, that not only is old smutted straw capable of conveying living spores to the soil at sowing time, but that they are also carried in manure from horses fed on such straw. Evidence has been obtained in Australia that wheat grown in soil in which a diseased crop grew the previous year, be-

comes infected even if treated with fungicides, showing that the spores shed on the soil retain their vitality until the next season. Hence, unlike bunt, where the spores that reach the soil otherwise than by



adhering to the grain coats usually germinate and die before the next crop is put in, flag smut persists in the soil and is capable of damaging a following wheat crop. The smut is often carried on the grain coats, but cleansing the grain is not enough by itself to protect the next crop, especially if stubble or manure, containing spores, has reached the soil.

The treatment is, therefore, more difficult than with bunt. Seed disinfection may not protect the crop in infected land. It should always be carried out, however, in order to kill any spores on the grain. The formalin treatment given under bunt may be used, the grain being sprinkled with the solution. There is nothing to gain by immersing the seed in the liquid, as there are no bunt balls to be removed. In addition, every effort should be made to avoid causing infection of the soil. In bad cases the stubble should be burnt. Care should be taken to avoid contamination through manure. In Australia, it is found necessary to pay particular attention to rotation in the affected localities and the practice of growing wheat only once in three years is becoming general.

The fungus does not seem to be capable of a saprophytic life after germination of the spores, and it is not yet certain whether any considerable proportion of the spores can go through such a prolonged high temperature with occasional rain as in the Punjab summer, without germinating and perishing. In Australia, moisture for some time before sowing is said to reduce infection from the soil, while if sown on the first rain, spores and wheat germinate together and there is much disease. Possibly in our climate seed treatment would give better results than in Australia.

A closely allied smut, *Urocystis occulta*, occurs on rye, but experiments have shown it to be distinct from that on wheat, neither being able to infect the host plant of the other.

**Mildew** (*Erysiphe graminis* DC.).--This is a disease of very minor importance in India, where it has only been found on wheat and a few wild grasses in the Himalaya and sub-montane districts of the north. Elsewhere it is common on a wide range of cultivated grasses and cereals, and in Europe it sometimes causes serious loss, especially to winter-sown grain crops. In Italy it has been found associated with the leaf-spot disease of wheat described below, the combined infection reducing considerably the yield.

The fungus develops on the leaves, usually on the upper surface, but sometimes also underneath, and on the sheaths and stems. It forms a

superficial, thick, flocculent growth, at first white, then grey or reddish. The young patches have a floury or powdery appearance from the formation of conidia of what is known as the *Oidium* stage of the fungus.

Later on, the spore-powder diminishes, and scattered black dots appear, partly immersed in the superficial growth. This is the perfect or ascigerous stage.



FIG. 57. Wheat mildew (*Erysipha graminis*): 1, part of affected plant,  $\times \frac{1}{2}$ ; 2, conidiophore and conidia from barley,  $\times 260$ ; 3, a perithecium; 4 ascus and ascospores,  $\times 260$ . (2 and 4 after Salmon, 3 after Delacroix).

tents of two specialised cells. The mature perithecia are about  $200\mu$  in diameter, globose-depressed, black, and partly immersed in the mycelium. Each consists of an outer envelope of brown pseudoparenchymatous cells, from the exterior of which short, simple or slightly branched, pale brown hyphae, known as "appendages", arise, and an inner cluster of 8 to 30 asci. The asci are cylindrical or ovate, 70 to 108 by 25 to  $40\mu$  in diameter and each contains 8 (rarely 4) elliptical, single-celled, hyaline spores, measuring 20 to 23 by 10 to  $13\mu$ . The ascospores are late in developing, but ripe ones may be got from old, dried-up plants placed in a moist atmosphere, or by immersing leaves bearing perithecia in water for some days. They are set free by the rupture of the perithecial wall which, in this family of the Ascomycetes, has no mouth.

Both conidia and ascospores can infect healthy wheat. They germinate readily in moist air, giving rise to one or more germ-tubes, which soon die unless they reach a suitable host plant. When, however, the spore germinates on wheat, the end of the germ-

The mycelium consists of superficial, sparingly branched, thick-walled hyphae, 4 to  $5\mu$  wide, which are interlaced into a web, covering a greater or less extent of leaf and stem. From this, short special conidiophores arise at right angles to the leaf surface. The conidiophore is swollen in a characteristic manner at the base, above which arises a long cylindrical outgrowth. The basal swelling is early cut off from the parent hypha by a transverse wall. The cylindrical outgrowth now divides into two cells, each of which again divides; and the four cells thus formed swell up into conidia, the junctions at the septa remaining narrow. They ripen from above downwards, the end spore being the first to mature. The basal cell next elongates in its upper part, below the chain of four conidia first formed, and new conidia are produced by the repeated division into two cells of the upper part of this elongation. The chain may consist ultimately of at least 10 or 12 conidia, which when ripe are elliptical, hyaline, single-celled bodies, measuring 25 to 30 by 8 to  $10\mu$  and falling off readily when disturbed.

The perithecial stage appears usually at about the same time as the ears. Each perithecium arises as the result of a sexual act, consisting in the fusion of part of the con-

tube forms a little swelling (appressorium) which becomes firmly attached to the surface and gives out on its under side a very minute, tubular outgrowth capable of penetrating the plant. Entry is effected directly through the outer wall of an epidermal cell; once inside, the tube swells up and develops a large haustorium in the cavity of the cell. The haustorium is elongated and has a bunch of finger-like projections from one or both ends, which serve to increase the surface through which food can be absorbed from the cell contents. With the support of the nutrition thus gained, the external portion of the germ-tube continues to grow, branches, and forms a mycelium. Other haustoria arise from this and penetrate the epidermal cells all over the affected part.

The effect of the parasite is to cause a discoloration and weakening of the plant, which is very noticeable in plants grown under shade, as in pot experiments. In bad cases the leaves become crinkled and often spirally twisted or deformed; the top of the shoot may wither and the ears, when formed, are light.

*Erysiphe graminis* attacks many grasses and cereals but is split up into specialised races (see p. 62), one of which is that on wheat. This race can only live on species of *Triticum* and, less readily, young plants of *Hordeum silvaticum*, which is not an Indian grass. Specimens sent from India have been tested in England and found to behave exactly as the European form in this respect. The forms on oats, barley, sugarcane, and other cultivated members of the grass family have not hitherto been found in India.

Treatment is not usually practicable. Sulphur dusting checks the parasite, as it does most of the Erysiphaceæ, but it is out of the question with a field cereal crop. It can be used, however, to protect experimental plants and was sometimes found necessary in such cases in Dehra Dun. Shade and a moist atmosphere encourage the spread of the fungus. The influence of certain manures on the disease is referred to on p. 123 above.

**Leaf spot** (*Leptosphaeria Tritici* (Garov.) Pass. = *Septoria Tritici* Desm. and *S. gramineum* Desm.).—This disease is common at Lyallpur and probably in other parts of the Punjab, but has not been found elsewhere in India. It has been described as causing much loss in several parts of Europe, sometimes alone but more usually associated with rust or mildew, so that it is difficult to decide which parasite is the true cause of the injury in these cases. In India it has been found alone and the damage was slight.

The fungus develops in the leaf tissues, forming elongated spots limited by the veins. The spots are at first yellow, then reddish-brown, and finally, when the tissues dry up, pale straw-coloured. In this last stage, numerous very minute, black dots, arranged in rows between the

veins, appear on both surfaces of the leaf. These are the fructifications (pycnidia) of the lower or *Septoria* stage.

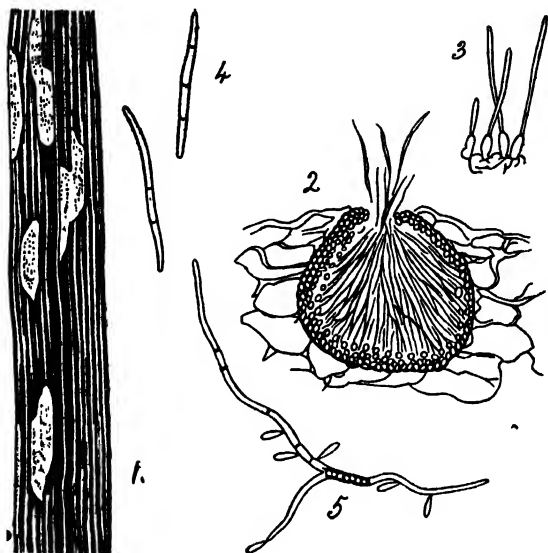


FIG. 58. Wheat leaf spot (*Leptosphaeria Tritici*): 1, spots on leaf showing pycnidia,  $\times 1\frac{1}{2}$ ; 2, a pycnidium in section; 3, young spores on their stalks; 4, ripe spores; 5, germination of spore with formation of secondary conidia. (2-4 after Prillieux, 5 after Krueger).

The pycnidia arise beneath the stomata of the leaf, as round, thin-walled, flask shaped receptacles, opening at the orifice of the stoma and filled with elongated, narrow spores. The pycnidia are about 100 to 200 $\mu$  in diameter; the spores, 30 to 75 by 1.2 to 2.5 $\mu$ , colourless and with 2 to 5 (generally 3) transverse walls. Germination occurs readily in water, a mycelium being formed which bears short secondary conidia. Infection takes place by the entry of the hyphae directly through the epidermis; the invaded cells are rapidly killed and, though spread is checked by the veins, leaves exposed to severe infection may wither completely. In extreme cases, the plants are stunted and ears are said not to develop or are sterile.

The pycnidial form has been artificially cultivated in Europe and gave rise to the ascomycetal fungus, *Leptosphaeria Tritici*, which had been previously observed to occur at times on the same leaves as the *Septoria*. This form has not been seen in India. It is also said that the fungus known as *Septoria gramineum* is probably identical with that under discussion. If all three are really the same, then the number of known host plants is considerable, and includes, besides wheat and oats, several wild grames. In India it has been found so far only on wheat.

The conditions which lead to a severe attack of leaf spot are much the same as induce rust. In dry, sunny weather the spores die in a few days. It is said that manuring with nitrate increases the susceptibility of the crop, while, on the other hand, sulphate of ammonia diminishes it. An application of basic slag at the time of setting of the grain has been recommended. Direct treatment seems as impracticable as in the case of rust.

**Mould** (*Mycosphærella Tulasnei* Jancz. = *Cladosporium herbarum* Lk.).—The conidial (*Cladosporium*) stage of this fungus is commonly found on withering green parts of plants in India and in many other parts of the world; indeed its spores are present so frequently in the air that it may be expected to appear on almost every bundle of plant specimens collected and kept without drying for a few days. Nevertheless its full life-history is not entirely understood, and in particular the question of its parasitism is still open to some doubt.

It is frequently found in India on the ears of cereals, particularly wheat, maize, and jowar, in moist localities or where the crop is so dense that aeration is deficient. It occurs less often on leaves and even stems of these and other plants, under similar conditions. Affected parts are covered with a greenish-black, mouldy growth. This is especially visible on ears of jowar and tassels of maize, which are sometimes turned black throughout whole fields. On wheat, the growth

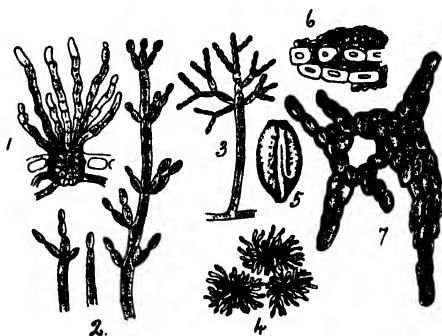


FIG 59. Wheat mould (*Cladosporium herbarum*): 1, conidiophores emerging from leaf; 2, tips of conidiophores with conidia, on left dwarf form, on right giant form; 3, conidial form known as *Hormodendron cladosporioides*; 4, conidial tufts from a leaf; 5, grain affected with mould; 6, section showing stromatic mycelium between the cells of the grain coats; 7, stroma more highly magnified. (4 after Prillieux, rest after Delacroix).

is not usually so dense nor the blackening so pronounced, except in lodged plants. When dry weather follows the infection, the superficial growth may be almost absent, the surface of the outer glumes being

smooth and dark brown in colour. In these cases the fungus often collects in the anthers and stigmas, and the pollen may be much reduced.

The effect on the leaves and stalks is to cause withering, followed often by a sort of wet rot, due no doubt to the invasion of the withered parts by saprophytic organisms. On the grain, brownish-black spots appear, accompanied sometimes by fissures which are occupied by the fungus growth. In wheat and jowar, the grain may be almost completely prevented from forming, the glumes enclosing nothing but the partially disorganised essential parts of the flower.

The mycelium is aggregated into numerous little greenish-black tufts, producing a velvety appearance on the surface of the ear. Within the tissues it tends to form layers of short-celled fungus parenchyma, which force their way down between the cells of the host. At and near the surface the hyphæ are greenish-brown; deeper in, hyaline.

Conidia are produced on tufts of erect conidiophores, which usually spring from little stromatic masses developed just below the epidermis. In this case, they frequently emerge through the stomata. Similar conidiophores can also arise from any part of the external mycelium and, in addition, the hyphæ themselves tend to break up when old, into spore-like articulations, the segments thus set free being capable of germinating. Typical conidiophores are greenish-brown, lighter in colour at the top, septate, and one or more times bent or kneed, each of the bends being capable of bearing conidia.

The conidia are formed in branched chains terminally and laterally on the conidiophores. The terminal chains pass insensibly into the conidiophore, the end articulations of which gradually take the shape of the spores. The first-formed conidia are often three or more in number, arising side by side on the end of the terminal cell of the conidiophore. Each of these may then bear two smaller conidia and each of the latter one or two more. The end spore of the chain is therefore the youngest. The basal conidia are 1 to 3-septate, most frequently 1-septate, while the terminal ones are usually continuous. The shape varies from cylindrical to oval or almost round; the colour from brown (older conidia) to nearly hyaline (young conidia); and the dimensions from 5 to 15 by 4 to 10 $\mu$ . Each conidium has a characteristic, thickened, refractive cushion at the ends, marking the point of union with the next spores of the chain. They fall off very easily, singly or in short chains.

A second conidial type (formerly known as *Hormodendron cladosporioides* Sacc.) is often associated with the normal form, and is considered by some to be the parasitic condition of the fungus. In this, the conidial chains are chiefly produced at the ends of a brush-like tuft of filaments formed at the tip of the conidiophore. The conidia are more numerous than in the *Cladosporium* form and are usually unseptate and almost hyaline. All sorts of transitional stages between the two forms occur; one gives rise to the other readily in culture; and it is probably more correct to describe the conidial fruit as being extremely variable than to consider them as representing two distinct stages in the life-history of the fungus.

The anisogamous stage (*Mycosphaerella Tularensi* Jancz.) has been discovered in Europe but has not yet been found in India.

The injury caused by this fungus results from the loss of grain when the attack is early, and from the depreciation of its quality when the attack is late. The mouldy grains make very inferior flour. In south





Fig 60 CROWN RUST (left) SMUT (middle) AND LEAF SPOT (right) OF OATS



India it is said that grains of jowar affected by the "wet weather mould," which is no doubt this fungus, are remarkable for their premature germination while still on the ear. How far the species is capable of infecting healthy, well-grown plants is not definitely known, and it seems probable that some of the diseases of plants such as melons and cucumbers grown under glass, said in Europe to be caused by this fungus, are really due to other parasitic species of *Hormodendron* or allied fungi.

No treatment is possible, the mould being, so far as most observations go, a symptom of unfavourable environment. Sowing in drills to promote aeration, and attention to drainage, are indicated in places where the disease habitually occurs.

### OATS (*Avena sativa* L.)

**Smut** (*Ustilago Avenae* (Pers.) Jens.).—Oat smut is known practically wherever oats are cultivated. In India, it is especially prevalent in Dehra Dun, where about one-tenth of the crop is annually lost from it, is common in the Punjab, and there have been several outbreaks in Bihar. Before treatment was adopted, it ranked with the most destructive diseases of crops in temperate climates, being estimated to have caused a loss of over £3,000,000 annually in the United States.

The spores are produced in the ovary and other parts of the flower, and also in the glumes and awns. The wall of the ovary persists as a distinct envelope around the spore-mass, for a considerable time, much longer than in the loose smut of wheat. Usually every head of a stool and all the grains of an ear are infected, but sometimes the upper grains escape. After the ear has fully opened, the membrane surrounding the mass of spores breaks up and the spores are blown off, ultimately leaving only the stalks of the spikelets behind.

The spores are nearly globose to oval, light olive brown singly, dark brown in mass, with one side less coloured than the other, finely spiny, and 5 to 9 $\mu$  in diameter. Germination takes place in a few hours in moisture, with the production of a 4 or 5 celled, stout promycelium. A sporidium is borne on each segment, usually near the septum but one is terminal. Sometimes a branch connects two segments, forming what is known as a buckle-joint, or two adjoining cells may unite by a clamp connection (p. 9). After the sporidia fall, they germinate by a fine germ-tube, or two become united by a short branch, through which the contents of one pass into the other, which then germinates. All these various unions are connected with the sexual reproduction of the fungus (see p. 26). In nutrient solutions, the sporidia bud freely after the manner of yeasts, and this period of saprophytic life may be prolonged by renewing the solution, for a year or more. The germinating power of the spores has been found to be retained at the end of 7½ years, unlike the loose smut of wheat where it is lost after a few months.

Infection only takes place in the young seedling, the greatest number of successful inoculations being obtained just after the primary shoot has emerged from the grain.

After the shoot is an inch in length, infection is rare. When oats germinate, the bud is pushed out of the glumes on an elongated base ("mesocotyl") which carries the primary node as much as  $\frac{1}{2}$  cm away from the origin of the primary root, in a week. This

elongated segment is short lived and infection takes place through it, below the primary node. Above the latter, infection is rare. The germ tubes only enter when the colls begin to collapse and die, at first they pass through the cells, later, as the sound tissues deeper in are reached, between them. They then turn upwards and pass above the primary node, through a narrow channel between the vascular tissue and the insertion of the first leaf. Here they begin to branch for the first time and the mycelium reaches the region of the growing point. Though the tissues below the primary node may contain many germ tubes few succeed in surmounting this barrier which becomes impassable as soon as its tissues are differentiated. But once the growing point is reached, the mycelium is probably never got rid of and is always able to continue its development up to spore formation.

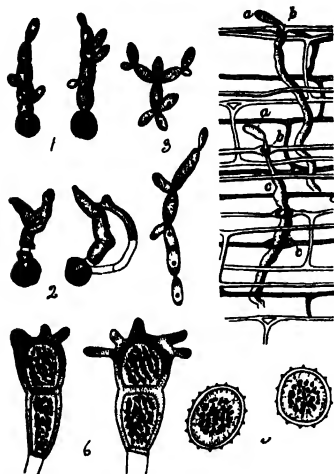


FIG. 61. Smut and rust of oats (*Ustilago Avena* and *Puccinia Lolii*). 1, smut spores germinating with promycelia and sporidia,  $\times 450$ , 2, fusion of promycelial cells,  $\times 450$ , 3, sporidia budding in nutrient solution,  $\times 350$ , 4, section of surface of young infected seedling, showing germinating sporidia a, the opening b through which the germ tubes penetrate, and the internal mycelium c,  $\times 350$ ; 5, uredospores of crown rust,  $\times 375$ , 6, teliospores of same,  $\times 500$ . (1-4 after Brefeld, 5-6 after Eriksson and Henning).

elongates, still producing no apparent effect on its host, until the formation of the ear. Then the hyphae accumulate in the young inflorescence as a preliminary to spore formation and, by their pressure, the host cells are gradually disorganised and killed.

Even after successful infection has been accomplished the oat plant may grow away from the fungus. The latter is only able to develop in young tissues and if the growing point elongates too fast may fail to reach it. The hyphae then break up into fragments and gradually disappear in the older tissues. Hence the oat plant is able to combat successfully a certain number of infections, and external factors such as temperature, humidity, and the like as well as varietal differences which influence the rate of growth of the seedling, have a similar effect in determining the severity of the disease to what they have in bunt. When the temperature at sowing time is low, oats are more likely to escape smut, since the spores require a considerably higher temperature for germinating than the oat grains themselves.

Infection may result either from spores present in the soil in a living condition when the crop is sown, or from spores adhering to the grain coat

and sown with the seed. The latter is much the more frequent and is, no doubt, favoured by the fact that smutted ears often open a little before the rest, so that the spores are set free and are blown about while the grain is maturing and before the glumes have closed around it. The dry, powdery spores of oat smut do not stick to articles with which they come in contact, so readily as those of bunt, and hence the danger of conveying the disease by infected sacks and implements is not so great.

Treatment by seed disinfection will, therefore, prevent the disease almost entirely. Several different methods are adopted, some being more popular in one country, others in another. Perhaps the most convenient is the formalin treatment, much used in the United States. This is employed as against bunt (p. 170), the sprinkled grain being kept moistened by the solution for four hours instead of two. There is no need to immerse the grain in the liquid, since there are not usually any smut balls unbroken. An experiment carried out in Dehra Dun in 1904-5 showed that formalin reduced the amount of disease from 1 plant in every 10. to 1 plant in every 1,700, without injuring germination in any way.

The hot water treatment is also used in the United States but is giving place to the last-mentioned method. It consists in dipping the grain in water at 132° or 133°F. for 10 minutes. It is advantageous to warm up the mass of grain first, by dipping the sacks or baskets in which it is contained into warm water at say 110° to 120°F. for a few minutes before transferring to the hotter bath. A thermometer must be used and a vessel of boiling water kept at hand so as to maintain the temperature at the right level. The grain should be stirred about, or if in a sack, lifted out, allowed to drain and plunged back, several times, to secure even heating. Properly carried out the crop may be entirely freed from smut, all the spores being killed.

Potassium sulphide is used in some countries in Europe, the grain being immersed in a solution of 1 lb. to 13 gallons water for 24 hours. The chief drawback to this method is that commercial potassium sulphide ("liver of sulphur") is of variable composition and often adulterated.

Copper sulphate, so effective against other smuts, is less satisfactory in the treatment of oat smut than the above remedies. This is because, owing to the way in which the spores are often caught between the glumes and the grain, a sufficiently prolonged treatment to be effective injures the germinating power of the seed. Thus in experiments at Dehra Dun, immersion in  $\frac{1}{2}$  per cent. copper sulphate for 24 hours was only half as effective as formalin in checking smut, while the crop was

decidedly inferior. One per cent. for the same period equalled formalin in efficiency but the crop was very poor. For shorter treatments, greater strengths have to be used and the injury to germination is equally great. A modification in general use in France, where it has the sanction of the Ministry of Agriculture, is as follows :—The grain is placed in a vessel to which is added enough of a solution of 1 per cent. copper sulphate to wet it thoroughly. Water is then added until there is a layer of about 6 inches of liquid over the grain, and after stirring well it is poured off with all light grains. After an hour, a second rinsing with fresh water is given and the grain then spread out and dusted thoroughly with slaked lime. It is turned over repeatedly until dry, when it is ready for sowing. In Germany, another method which gives the same results is practised. The grain is immersed for 12 to 16 hours in  $\frac{1}{2}$  per cent. copper sulphate solution, then taken out and plunged in milk of lime of 6 per cent. strength, for 5 minutes, after which it is spread out to dry. In both these cases, the lime neutralises the caustic effect of the copper sulphate and prevents the injury caused by this compound to the delicate primary shoot of the seedling. Further, a certain amount of copper carbonate is left adhering to the grain and appears to offer some protection against the attack of spores pre-existing in the soil.

Except the last, none of the above methods guard against infection from spores in the soil, but although it has been proved that the saprophytic budding stage of the sporidia may persist for at least a year under exceptional conditions, in practice it is not found that any considerable amount of smut results from this source. One or other of the treatments given above is universally relied on to reduce the loss to a negligible quantity.

As with bunt, cases have been recorded where the use of heavily smutted oats as fodder has proved detrimental to the health of farm animals, especially in North America. The evidence is not conclusive but there seems to be a certain amount of risk of even fatal results.

The wild oat (*Avena fatua*, L.) is sometimes attacked by this smut in other countries.

**Rust** (*Puccinia Lolii* Niels. = *P. coronifera* Kleb.).—In most cereal producing countries oats suffer at least as severely as wheat from rust. In India, however, the black rust (*P. graminis*) has not yet been found, though so common on wheat and barley, while the crown rust (as *P. Lolii* is called) has only once been recorded, at Pusa in 1907. Indian oats have been introduced into several countries in the hope that they might prove

immune to rust, but this hope has not been realised, and indeed in the Transvaal they were found to be highly susceptible to both black and crown rusts.

The appearance of a plant attacked by crown rust is shown in Fig. 60 and the characters of the spores in Fig. 61. Since the disease is not at present established in India, it is not necessary to do more than illustrate it, so that it may be recognised if met with.

**Leaf spot** (*Helminthosporium Avenæ* Br. and Cav.).—The leaf spot of oats is very common in India, especially on young plants. In Pusa, it appears every year when the seedlings are a few inches high and persists up to the time of harvest. The damage caused is probably not great, but it is very difficult to estimate it.

The appearance of an affected leaf is shown in Fig. 60, the leaf blade being marked by numerous yellow spots with a brown ring towards the centre. In severe attacks, the leaf dries up in long streaks.

The spots are due to the mycelium of the parasite, which ramifies between the leaf cells and causes the destruction of their chlorophyll. No haustoria are formed but the invaded tissues collapse and dry up rapidly.

Sporos are produced on the old spots only. The conidiophores arise from the internal mycelium and emerge chiefly through the stomata. They are brown, simple, septate, erect hyphae, often with knee-bends, especially towards the free end; these are due to the conidia being formed terminally and then pushed to one side as the conidiophore continues to elongate. Each bend on a fully-grown conidiophore thus represents the position where a terminal conidium was formed at an earlier stage, (Fig. 64, 5)

The conidia are deep brown, long, cylindrical bodies measuring 80 to 100 by 15 to 16 $\mu$  and with a variable number of septa, usually 4 to 6. They germinate readily in water, the germ-tubes being able to penetrate the tissues of the first green leaf without difficulty. Entry is through the epidermal cells, not by the stomata.

It is known that in the allied disease of barley, caused by *H. teres*, the attack in many cases results from infection of the grain or the young seedling, at the moment of germination, by means of spores or mycelium present on the glumes at the time of sowing. Conidia are developed on the first leaf from this primary attack and serve to cause a generalised secondary attack, when the climatic conditions are favourable. If this also occurs in oats, it is probable that the seed treatment recommended for the barley disease (p. 190), would be effective in checking the primary attack. The disease would not, however, be appreciably reduced unless the treatment was carried out on a sufficiently large scale to prevent secondary infection from neighbouring untreated crops.

**BARLEY** (*Hordeum vulgare* L.).

**Covered smut** (*Ustilago Hordei* (Pers.) Kell. and Sw.).—Scattered cases of this disease occur in the barley fields all over northern India, but never to the same extent as oat smut. It is known almost everywhere that barley is cultivated. A second smut—naked smut (see below)—is also widely distributed in other countries, but has only rarely been seen in India.

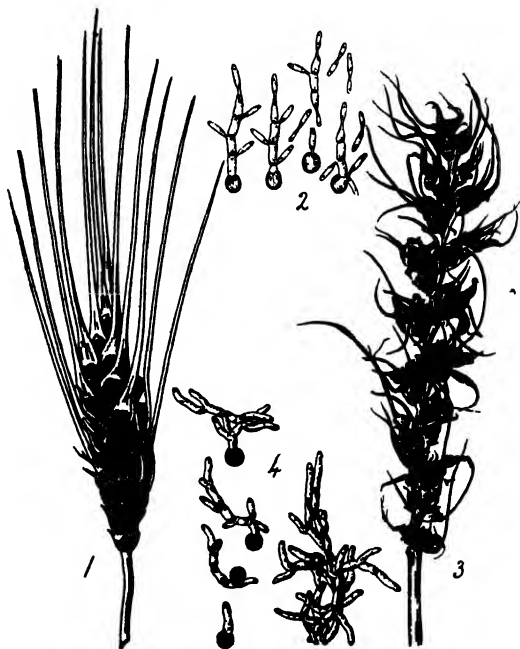


FIG. 62. Smuts of barley (*Ustilago Hordei* and *Ustilago nuda*): 1, ear with covered smut, nat. size; 2, germination of spores of same with promycelia and sporidia,  $\times 230$ ; 3, ear with loose smut, nat. size; 4, germination of spores of same with branching germ-tubes and no sporidia,  $\times 230$ . (2 and 4 after Brefeld).

There is nothing to distinguish affected plants from sound ones, until the ear is formed, when the spore-masses develop in the ovary of the flower, forming a black, compact body, enclosed by the ovary wall and glumes. The affected ears may remain more or less enclosed in the leaf sheath.

The spores tend to remain in a compact mass, though not so firmly adherent as in bunt. Individually they are round to ellipsoidal, brown, often lighter coloured on one side, quite smooth, and 6 to 9 or sometimes up to  $11\mu$  in diameter.

Germination occurs in water or damp soil, even after several months. A three-septate promycelium is formed which bears sporidia terminally and laterally. Buckle-joints are common, as in oat smut. The elongated sporidia continue to bud off fresh spores after falling, and in nutrient solutions this saprophytic life can be prolonged as long as fresh nutrition is supplied.

The life-history is similar to that of oat smut, infection taking place in the seedling stage and the fungus growing up inside the plant until the inflorescence appears, when spore formation begins. Dissemination by the wind does not occur so readily as in oat smut, owing to the persistence of the plant tissues enclosing the spore-mass.

The treatment is the same as in oat smut. When, however, the loose smut is also found in the crop, the hot water treatment must be used as described below.

**Loose smut** (*Ustilago nuda* (Jens.) Kell. and Sw.).—This is much rarer than the last in India, which is fortunate as it is more difficult to control. It resembles the loose smut of wheat (p. 163) in all important respects.

The sori develop in the spikelets, enclosed by a thin, silvery membrane which ruptures as the ear emerges and sets free the loose spore-mass. The latter is soon dissipated, leaving the naked rachis behind.

The spores are nearly round, 5 to  $9\mu$  in diameter, with a light brown wall, paler on one side than the other, and with faint spines. They germinate by forming a branched germ-tube without sporidia, as in *U. Triticis*, and like the latter are not very long-lived. Infection occurs through the flower, just as in loose smut of wheat.

The hot water treatment is that most frequently used (p. 165). As barley grain is less resistant to heat than the other cereals, a temperature of  $126^{\circ}\text{F}$ . only is allowable for the hot water bath, but the grain may be left in for 15 minutes, by which time the fungus will have been killed.

**Rust** (DWARF RUST (*Puccinia simplex* (Koern.) Eriks. and Henn.) BLACK RUST (*Puccinia graminis* Pers.), YELLOW RUST (*Puccinia glumarum* Eriks. and Henn.).—Barley, like wheat, is subject to the attacks of three distinct rusts, two of which are the same as two of the wheat rusts, while the dwarf rust of barley takes the place of orange rust of wheat.

Black and yellow rusts are common and quite similar to the forms on wheat. They are specialised on the two hosts, those on barley being unable to pass to wheat, while those on wheat can attack barley decidedly less readily than its own forms. In Europe, the same results have been obtained, except that there the yellow rust of wheat is said not to attack barley at all. In South Africa, black rust from wheat attacks barley readily in summer, but not at all in winter. It is worthy of note that neither from wheat nor barley can black rust pass to oats in India, though in Europe the black rust of wheat appears to be able to attack oats and in the United States also will do so, if first passed through barley, though unable to infect directly.

Dwarf rust is less common than the others in India, having been but rarely seen. It is confined to species of *Hordeum*.

The uredo sori are small, citron-yellow in colour, and irregularly scattered on both surfaces of the leaf, resembling somewhat those of *Puccinia triticina*. The uredospores are roundish or ellipsoidal, yellow, 19 to 22  $\mu$  in diameter when round and 22 to 27 by 15 to 20  $\mu$  when elongated. The wall is rather thick, spiny, and with numerous germ-pores.

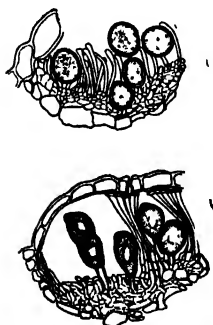


FIG. 68. Dwarf rust of barley (*Puccinia simplex*): part of uredo sorus (above) and teleuto sorus (below),  $\times 260$ .

The teleuto sori are scattered as small, black crusts on the leaf but are more elongated on the leaf sheaths and stem. They are long covered by the epidermis and often confluent. The teleutospores are oblong-clavate or pear-shaped, the apex being rounded, flattened, or drawn out to one side, and the wall thick and usually broader near the apex than lower down. Unicellular and bicellular spores occur in the same sorus. They are chestnut-brown in colour, smooth-walled, and 25 to 54 by 15 to 24  $\mu$  in diameter, the single-celled spores being the smallest. The stalk is short and brownish. The sorus is usually divided into compartments by clusters of brown paraphyses, which spread out and become flattened under the epidermis. These, and the numerous single-celled teleutospores ("mesospores") enable the species to be readily identified.

It is probable that the barley rusts agree with those of wheat in their dependence on climatic conditions, date of attack, and other general characters, and the section under wheat rust should be referred to for further particulars.

**STRIPE DISEASE** (*Helminthosporium gramineum* Rabenh.).—This disease is said to be increasing in Europe, and is also known in the United States, east Asia, and other parts of the world. In England, it is sometimes called "blindness" or "deaf ear," one of the most marked symptoms being the failure of the ear to set its grain properly. Losses of 20 to



25 per cent. have been recorded in Europe, but in India and in the United States such serious damage has not yet been noticed.

The disease begins by the appearance of small, pallid spots on the leaves and sheaths; these may be visible on the first leaf of the seedling or not show until the plant approaches maturity, but when once a leaf is affected, usually all the succeeding leaves are similarly attacked. The spots early extend to long, parallel streaks, which may be continuous from tip to base of the leaf, or be broken by bands of healthy tissue. As development proceeds, they turn brown and the whole leaf gradually dries up. Old spots usually have a brown margin, with a straw-coloured centre in which the spore-clusters appear as greyish-black patches.

The effect on the plant is considerable. In many instances the ear does not emerge from its sheath; if it does, it remains erect and stunted and with the awns often twisted and bent (Fig. 64, 2). Even when

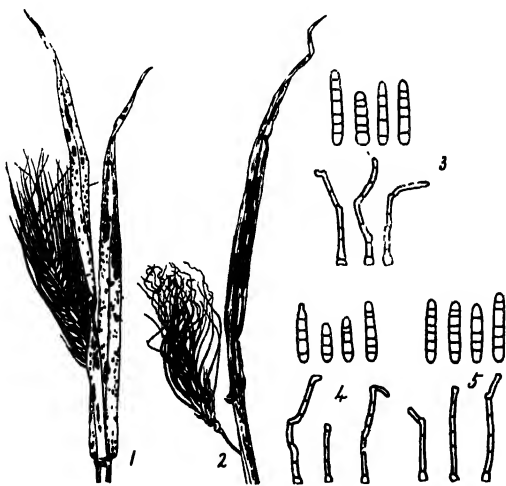


FIG. 64. *Helminthosporium* on barley and oats: 1, late blight of barley (*Helminthosporium teres*); 2, stripe disease of barley (*Helminthosporium gramineum*); 3, conidiophores and conidia of *H. teres*,  $\times 100$ ; 4, same of *H. gramineum*,  $\times 100$ ; 5, same of *H. avenae*,  $\times 100$  (3-5 after Kölpin Ravn).

formed, the grain does not mature properly, but remains small and soft. The leaves often split or shred, and wither early; every shoot of the stool is usually affected; and growth is checked so that the plant is often only half the normal size.

The hyphæ are found between the cells of the discoloured tissues. They give off clusters of thick, erect conidiophores, which emerge through the stomata and bear spores (Fig. 64, 4). There is no constant difference between the conidial stage of this fungus and that of *Helminthosporium Avenæ*, described on p. 183 above, except that in the latter the conidiophores are usually single, not clustered.

In Europe, the fungus is said to have a further stage, which has been obtained in artificial culture and observed sometimes also on the plant. Masses of dark hyphæ unite to form black sclerotia, composed of pseudoparenchymatous cells and furnished with a covering of stiff, black hairs. Each of these sclerotia may, under favourable conditions, develop a single perithecium, containing asci and spores. The latter are elliptical, with three transverse septa and often also a longitudinal one, brown in colour and, when young, with a gelatinous sheath around the spore. They measure 44 to 54 by 17 to 20 $\mu$  in diameter. This stage of the fungus, which has been described as *Pyrenophora (Pleospora) trichostoma* (Fr.) Sacc., has also been obtained recently in the United States, where, however, it is said to belong not to the present species of *Helminthosporium* but to that next to be described. It has not been seen in India.

Examination of the growing point of diseased plants shows that the hyphæ can be found between the young cells of this part, just as in smut, and they reach the leaf by passing out into the young leaf-buds. It has been proved that the disease originates in the majority of cases from contamination of the seed prior to sowing, by conidia which adhere to the grain at the time of ripening. Some cases also probably arise from direct infection of the seed by the internal mycelium in the growing point of the shoot, or come from infected straw bearing sclerotia or perithecia in the soil. Secondary spread from the first-formed conidia is probably rare. Hence the disease resembles the covered smut in many respects and it is easy to understand why all the shoots of a stool, and all the leaves formed after the first which shows the spots, are usually diseased.

The treatment is, therefore, similar to that of covered smut or the smut of oats. The best results have been obtained in some cases with formalin an increase of yield of 25 per cent. having been noted. With copper sulphate, it has also been practically prevented, but the germination of the treated grain was reduced by about 15 per cent. The hot water treatment is recommended where naked smut is also present, as it is the only treatment that checks both diseases. Recently, what is known as the intermittent hot water treatment has been advocated, as the ordinary hot water bath at 126°F. is said sometimes to injure the grain seriously. The seed is put in sacks about three-fourths full, and placed in water at about 104°F. for 3 hours, then in water at 118°F. for 10 minutes, then dipped again for about a minute in the bath at 104°F. and left for 2 hours in a heated room. Finally it is given another 10 minutes at 118°F. and spread out to dry. This complicated method could scarcely be used outside experimental farms.

Late-sown crops are said in Europe to escape injury at times, but there is no evidence to show what effect late sowing would have in India.

The fungus also attacks certain wild grasses in Europe and may be found on other hosts in India, on further search.

**Late blight** (*Helminthosporium teres* Sacc.).—The parasite which causes this disease is practically indistinguishable in its conidial stage from that last described, but its effects on the plant are quite different. It is considered to be the worst disease of barley in parts of the United States and is also common, but apparently less destructive than stripe disease, in Europe.

The leaves become covered with irregular, oblong or elongated, dark greenish-brown spots, visible on both surfaces. Similar spots occur also on the glumes and spikelets. There is no deep-seated alteration in the growth of the plant, except such as occurs from interference with the functions of the leaves. The latter may turn brown and wither but do not split into shreds as in stripe disease, and although the loss of grain may be considerable, the ears are not distorted in the same way as in the last case (Fig. 64, 1). In bad attacks, the straw is dull brown with brick-red spots, and instead of standing erect at harvest time, falls over in a tangled mass.

All stages of attack may be seen on a single plant; the basal leaves may be quite dried up, those above progressively less spotted, while those at the top may be clean. Spread is rapid and, unlike stripe disease, the infection may quickly run through a whole field.

The hyphæ are found between the cells, as in the last species. The conidiophores come out in clusters from the stomata and are reddish or dark brown, septate hyphæ, up to  $180\mu$  long, with a somewhat flexed, blunt tip, on which the conidia are borne singly. The latter are straight or slightly curved, 100 to 150 by 15 to  $20\mu$  in diameter, widest in the middle, greenish-grey in colour (grey-black when old), and with 7 to 14 septa (Fig. 64, 3). After they fall, new ones may be borne at the same place. Germination occurs by germ-tubes from the end cells, and occasionally one or two from the middle cells. New conidia may appear in culture in a few days or even, at about  $25^{\circ}\text{C}$ ., in 24 hours.

Sclerotia are formed as in *H. gramineum*, and give rise to pycnidia containing thin-walled spores, 2 to 4 by 1 to  $1.5\mu$  in diameter, embedded in mucus. According to workers in the United States, it is these sclerotia, and not those of *H. gramineum*, that ultimately bear the perithecia of *Pyrenophora trichostoma*, referred to under the last fungus.

Infection occurs from mycelium located in the glumes; from conidia on the grain or in the soil, the germ-tubes penetrating directly through the epidermis; from sclerotia in the soil or on dead straw; or from the pycnosporos and ascospores borne in these sclerotia. In direct inoculations on the grain, it was found that the attack was so severe that many seedlings

failed to emerge or died down soon after they appeared. Those that grow beyond this stage may show the first spots when about 8 inches high or even sooner. These come from an internal mycelium which has grown up inside the young shoot, but there is no generalised infection of the plant as in stripe disease and subsequently-formed leaves or shoots may escape attack, the internal mycelium failing to reach them. This appears to be due to the fact that the growing point remains free from the fungus. On the other hand, the conidia produced on the first spots readily infect other leaves to which they are blown, and this accounts for the rapid spread of the disease from plant to plant.

Hence two types of attack can be distinguished: a primary, due to infection of the seed; and a secondary, due to infection from the conidia borne on the primary spots.

The attack varies in severity according to the variety, two-rowed barley being generally less damaged than six-rowed. The time of sowing is also important, as the parasite does not develop freely at low temperatures, which are not low enough to prevent barley from germinating. Secondary spread by conidia is closely dependent on the weather conditions during the growth of the crop.

Seed treatment has been successful in checking this disease in the United States. Copper sulphate is effective, but has been found, as usual, to injure germination; good results have, however, been obtained by soaking in  $\frac{1}{2}$  per cent. for 14 hours, followed by dipping in milk of lime (see under oat smut). Formalin of a strength of  $\frac{1}{2}$  per cent. or a little more (1 part to 160 or 200 parts water) is recommended in the United States. The hot water treatment is also said to be satisfactory, the grain being first soaked in water at ordinary room temperature for 4 hours and then plunged in hot water at 126°F. for 15 minutes (see under loose smut of wheat). It must be remembered that barley is more susceptible to heat than the other cereals, and even this treatment is said in Europe to cause injury; the complicated intermittent hot water treatment introduced to replace it has already been referred to under stripe disease.

It has been pointed out in Europe that though seed treatment will check the primary attack, it cannot prevent secondary infection, by conidia, if even a few primary infections escape the treatment. The crop can also be infected from neighbouring untreated fields. Hence, to be successful, it must be carried out on the whole crop, and this is probably why it seems to have been, on the whole, less effective in practice than against stripe disease.

**MAIZE (*Zea Mays* L.).**

**Downy mildew (*Sclerospora Maydis* (Rac.) Butl.).**—The destructive disease caused by this fungus was first described in Java in 1897, when it was predicted that extension to other maize-growing countries would probably occur. It was first seen in India at Pusa, in 1912, and was certainly of recent introduction, but when it reached India is unknown. In Java it causes epidemics of great severity, but at Pusa the damage has been slight up to date. It is known also in the Philippines.



**FIG. 65.** Downy mildew of maize (*Sclerospora Maydis*). Effect on plant.

The disease becomes apparent before the plants have attained their full growth. The upper part of affected plants turns whitish, owing to the disappearance of the leaf chlorophyll in long streaks, the lower leaves remaining normal. The growth of the plant is checked and the internodes frequently shortened, so as to give a bunched appearance to the head (Fig. 65). Single plants only are attacked, there being no evidence of spread from plant to plant, and the scattered cases of disease can easily be picked out by their pale colour and stunted, bunched growth. Such plants usually produce no grain (though sometimes small cobs are borne) but the tassel is not uncommonly formed in a normal manner. The whole plant withers and dies, as a rule, some weeks before the rest of the crop is ready for harvest.

The parasite is often visible as white, downy or woolly patches on both surfaces of the pale streaks. These are composed of the conidial stage of the fungus.

The hyphae are immersed in the leaf, where they pursue a tortuous course between the cells of the leaf mesophyll in the discoloured areas. They are large, unseptate, and with lateral swellings which indent and perhaps penetrate the cell walls, acting probably as haustoria. When fructification is about to begin, hyphae collect in the sub-stomatal air spaces, and from them small clusters of conidiophores emerge through the stomata on both surfaces of the leaf.

The conidiophores are very thick and rather short, being usually about 20 to 25  $\mu$  in breadth and 150  $\mu$  long, the length, however, varying considerably. They are usually unbranched at the base, but fork twice or three times near the tip, the end branches being stout and provided each with two or more sterigmata. Each sterigma bears a single conidium, which falls off when ripe, leaving the conidiophore devoid of spores.

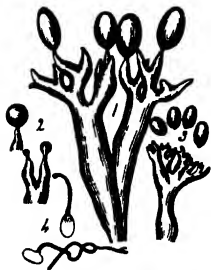


FIG. 65. *Sclerospora Maydis*  
1, conidiophores with ripe conidia,  $\times 210$ ; 2, early stage in conidium-formation,  $\times 210$ ; 3, conidiophore with fallen spores,  $\times 110$ ; 4 germination,  $\times 110$ .

The conidia are spherical when young but broadly oval when mature, hyaline, thin-walled, not papillate or stalked, and 25 to 45 by 16 to 22  $\mu$  in diameter. They germinate readily in water, and may be found germinating in large numbers on infected leaves in the field. One or two germ-tubes arise from any part of the spore, giving sparingly branched, rather slender hyphae, often of considerable length. The subsequent history is unknown, as the fungus has not been cultivated and no successful inoculations from conidia have been obtained. Germination by zoospore has never been observed.

Unlike the allied species, *Sclerospora graminicola*, common in India on jowar and other plants, the second or zoospore type of fructification has not been found, in spite of repeated search. It is quite unknown how

the fungus persists from year to year or how infection occurs, though the characters of the disease suggest generalised infection from the seedling stage, as in oat smut, and recently the hyphæ have been detected close to the growing point of affected stems at Pusa, just as in the latter disease. Indeed, though oospores have been found in enormous numbers in some of the allied species, they have never been germinated, and the life-history of the members of this genus (which only occurs on cereals and other grasses) is the most obscure amongst the Peronosporaceæ. Of recent years they have been found on many cereals (wheat, oats, maize, jowar, bajra, Italian millet, rice), and on sugarcane, in various parts of the world, and cause a group of diseases of considerable (and apparently increasing) economic importance.

The treatment, on the information at present available, should be directed to preventing the persistence of the parasite from the previous crop. For this purpose it will probably be sufficient to remove, and destroy all diseased plants before they wither. Such plants can easily be recognised in the field and, so long as the disease remains sporadic, there should be no special difficulty in carrying out this measure. Until the life-history is better understood, rational treatment cannot be recommended.

The closely related fodder plant, *Euchlæna mexicana*, is attacked by a similar disease in India, but the parasite has been provisionally referred to *Sclerospora graminicola*, in the absence of the oosporal fruit. It may prove, on further study, to be identical with the above.

**Rust** (*Puccinia Maydis* Bérang.).—This rust is fairly common in Bombay but appears to be infrequent elsewhere, and does not occur in the extensive area under the crop in Bihar. It is most abundant under conditions of relatively high temperature, as in South Europe, the warmer parts of the United States, Africa, and Australia.

The damage is rarely considerable in India, though it is said to do harm at times in other countries, especially in thickly-planted maize during periods of high humidity. Under these conditions, the leaves may become covered with innumerable uredo sori, lose their green colour, droop, and dry up, and the plant may fail to form its cobs.

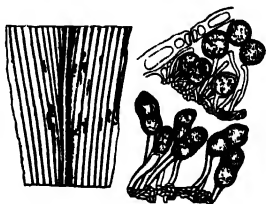


FIG. 67. Maize rust (*Puccinia Maydis*).—part of a leaf with sori,  $\times \frac{1}{2}$ ; and spore details,  $\times 200$ .

The uredo sori appear scattered or in groups on both surfaces of the leaf, at first as little yellow points, which elongate (sometimes becoming confluent), turn reddish-brown, and become pustular. The pustules burst soon, disclosing the reddish-brown, powdery spore-mass. The teleuto sori appear later, as larger black patches, up to 2 mm. long and sometimes confluent. They remain long covered by the epidermis but burst eventually.

The uredospores are elliptical or ovoid, light brown, finely spiny, and measure 24 to 32 by 20 to 28 $\mu$  in diameter. They have 4 germ-pores. Infection occurs as in the wheat rusts, the sub-stomatal vesicle being triangular in shape in longitudinal section. Two or more infection hyphae arise from the basal angles. The ordinary hyphae are large (4.5 to 5 $\mu$  across) and send branched haustoria into the leaf cells.

The teleutospores are oblong or ovate, sometimes rather pointed at the apex, which is thickened, constricted at the septum, and bright chestnut-brown in colour. They measure 28 to 48 by 13 to 25 $\mu$  and are borne on rather long, persistent, brownish stalks which are thickened towards the top.

Until recently this species was thought to have no aecidial stage. It is now known, however, that it is heteroecious, like *Puccinia graminis*, the aecidium being formed on species of *Oxalis* in America, Europe, and South Africa, where it has been known for many years as *Aecidium Oxalidis* Thum., *A. Peyruschianum* Magn., etc. It has recently been established that this aecidium can be obtained by inoculating *Oxalis* leaves with sporidia from the teleutospores of *Puccinia Maydis*, while by sowing aecidiospores from *Oxalis* on maize, the maize rust can be produced. As in the wheat rusts, however, there is no doubt that the aecidial stage (which has not yet been seen in India) can be dispensed with.

A second maize rust is known in South Africa, where it is distinguished as red rust, the present form being called brown rust. The red rust is attributed to *Puccinia purpurea*, which is common in India on jowar (*Andropogon Sorghum*) but has not been seen here on maize. The only other grass known to be attacked by *Puccinia Maydis* is the American fodder-grass, *Euchlana mexicana*, a very close ally of maize.

As the disease is seldom epidemic, it has not been as fully studied as the rusts of other cereals such as wheat, and no suggestions for its control have been made.

**Smut (*Ustilago Zeae* (Beckm.) Ung.).**—The common smut of maize is one of the most remarkable of the diseases caused by the Ustilaginæ, on account of the striking tumours which it causes on infected plants. It occurs in most countries where maize is cultivated. In India, it is confined to the north-west, being prevalent in Kashmir, less common in the Punjab, rare in the United Provinces, and unknown in Bengal and Bombay. Except in the temperate parts of the north-west, the damage done is slight. In Kashmir, it is considerable, the total loss to the maize crop from this and the next smut having amounted to an appreciable proportion of the harvest in 1908. In other countries the losses vary greatly from year to year. In parts of the United States the variation may be from 0 to 25 per cent. In Europe, losses of 20 per cent. were



observed in many parts of Hungary in 1902-03, while up to 30 per cent. has been noted elsewhere ; in 1847 the crop was ruined in parts of the south of France, and in 1879 much of the harvest was destroyed in places in Italy. Generally, however, the damage is much less than this in Europe and the disease is only sporadic in ordinary years. The climatic condition which most favours its appearance is said usually to be damp, warm weather during the period of most vigorous growth of the crop. In America, however, it is sometimes said to be prevalent in dry years.

The disease appears in the form of soft tumours, on any part of the plant, from the roots to the tassel and cob. Most commonly the cob is affected, least often the roots and then chiefly the adventitious roots above

ground. Seedling plants are difficult to infect ; if attacked they are either killed or stunted, but in the latter case may mature quite healthy ears. Usually the first boils are seen on the leaves when the plant is more than a foot high ; they are generally small and



FIG. 68. Maize smut (*Ustilago Zeæ*) : affected cob,  $\times 2$  ; spores and their germination,  $\times 450$ .

wrinkled, at first white, then dark from the development of spores in their interior. As the plant grows, further tumours appear, especially at the junction of the leaf sheath and blade, and at the nodes of the stem. Here they may reach a very large size, stem tumours as large as a child's head being sometimes found. When the tassel appears, small pustules are often found in the male flowers, each individual organ of the flower being converted into a smut boil. The female inflorescence, or cob, is very frequently attacked, the whole ear, or more often only individual flowers, being affected. The ovary may remain rudimentary or aborted, or may be hypertrophied to a tumour the size of a nut ; around it the floral scales are swollen, sometimes forming flattened masses,

sometimes elongated to an inch or more and often quite hiding the ovary. Sometimes the large spathes enclosing the cob are affected by tumours of considerable size. Even when only a few of the flowers are attacked in a cob, the amount of grain produced may be greatly reduced, as the grains near the smut tumours frequently fail to develop.

The structure of the young tumour is of interest. It varies somewhat according to its localisation and the age of the tissues, but the general result is the formation of a tissue rich in nutrient material, to serve as a food supply for the parasite. There is a rapid hypertrophy, due to the development of a tissue of thin-walled parenchyma between the epidermis and the outer bundle sheath. The size of the elements in the bundles and sometimes also in the ground tissue is diminished. The bulk of the tumour comes to consist of nutrient parenchyma, rich in starch and other cell contents and with large nuclei; this is penetrated in all directions by small bundles, composed largely of sieve tubes and procambial cells, the latter filled with dense plasma. The water-carrying vessels of the xylem are not developed, except at the margin of the tumour, and the xylem elements remain unmodified. In the bundle sheaths, the cells multiply and remain thin-walled, while later on, masses of thin-walled parenchyma, rich in starch, are found within the bundles. The ultimate ramifications of the latter are composed of short, narrow, thin-walled, cylindrical cells, placed end to end, and not differentiated; these are filled with a granular substance and have large nuclei.

The nutrient tissue is entirely used up during the process of spore-formation of the fungus, and the ripe smut boil consists of a mass of spores, mixed with which are the remains of the emptied and collapsed parenchyma cells of the host, while the persistent bundles ramify as a fibrous network between the spores. The epidermis, which covers the tumour as a thin, white membrane, differs from the normal leaf epidermis in being composed of thin-walled, uncuticularised, polygonal cells, with few stomata, many of which are distorted, and without hairs. It ruptures easily and permits the spores to be disseminated into the air.

Deep-seated alterations may be caused in the inflorescence as a result of the attack. It is well known that in maize the male and female inflorescences are separate, the former terminal (the "tassel"), the latter lateral (the "cob"). In smutted plants, however, it has sometimes been observed that the male inflorescence bears female and hermaphrodite flowers in its lower part and may even give perfectly normal grain at the base.

Each individual tumour arises from an independent infection at the locality where it is formed. Infection may take place at any part of the plant where growing tissue is present but cannot occur once the cells have ceased growth and reached maturity. Hence the earlier infections are usually on the leaves, the only growing parts visible when the plant is young. As growth continues longest in the leaves at the junction of the sheath and blade, this part is exposed to infection after the rest of the leaf has become immune. When the elongating stem becomes visible, especially at the nodes, stem tumours begin to appear. Then the tassel comes out and in turn becomes liable to infection. Last of all, the cobs

begin to form, and as the spores produced earlier, on other parts of the plant, are now mature, infection of the cobs is naturally very likely to occur. It has been found in the United States that infection of young tissues is assisted when they are wounded; it is stated that the attacks of boring insects in such localities are often followed by the formation of smut tumours, and it is suggested that insects may be an important agency in bringing about infection. Plants of low vitality are also easily attacked, and rapid total infection can be secured by exposing the plant to the vapour of ether, or by heating it to 70°C. As the parasite lives on surplus food stored in the nutrient tissue by the plant, its growth can be accelerated by reducing the effort required by the plant to provide this food, as for instance by giving sugar to the plant.

Infection occurs by the entry of a germ-tube from a sporidium which has lodged on any actively growing part. The germ-tube bores through the young epidermis and at first grows into and through the cells of the parenchyma below, stimulating them to active division. Some of the branches extend rapidly throughout the tissues, so as to spread the infection over a considerable area; these are known as infection hyphae. Others form little clumps, with many branches, within individual cells; these are the feeding hyphae, which may be compared to the haustoria of parasites that live mainly between the cells, as the rusts of wheat. The infection hyphae may either grow between the cells, sending feeding hyphae into them, or may pass directly across a cell. In the latter case, the fungus does not, as a rule, come into contact with the cell contents, being enclosed in a cellulose tube or sheath which is manufactured around the hypha the moment it enters the cell, and keeps pace with its growth. This sheath is developed through the activity of the cell protoplasm, and is a defensive device by means of which the cell endeavours to protect itself against the injurious action of the fungus. Sometimes it is formed so rapidly and in such strength as to stop the growth of the hypha altogether. In the young, thin-walled parenchyma of the newly-formed tumour, the bulk of the fungus is found chiefly between the cells, and up to the time of spore formation there is no destruction of the cell contents, the parasite living on surplus food supplied by the plant. The infection hyphae, passing across the cells enclosed in their cellulose sheaths, are best seen towards the margin of the tumour. The hyphae do not confine themselves to the cells of the parenchyma but also penetrate the young bundles, being found, for instance, in the large vessels of the axis of the inflorescence in diseased ears. They do not, however, extend far beyond the seat of the primary infection, being limited to the swollen tissues of the tumour. The infection is thus local in maize smut, in contradistinction to such cases of generalised infection as oat smut and the stripe disease of barley.

When the parasite reaches maturity, about three weeks after the infection, dense clumps of hyphae collect within the cells here and there throughout the tumour. The ends of these hyphae are richly branched and segmented into short, uninucleate lengths. Soon the wall between each pair of segments dissolves and an irregularly cylindrical cell with two nuclei results. The wall of this becomes softened and gelatinised, the cavity of the hypha almost disappearing, and as the neighbouring hyphae and their branches coalesce by their softened walls, their individuality is in great part lost. The swollen mass then becomes differentiated into numerous binucleate portions, surrounded by a dense gelatinous sheath. These are the spore origina, at first irregularly polygonal from mutual pressure, but soon almost spherical. Each spore origin consists of a protoplasmic core with two nuclei and oily contents, surrounded by a gelatinous wall. The latter soon becomes more definite in contour, and a dark line appears in it at a short distance from

the outer edge. This becomes the outer spore wall or exospore, which is provided with fine spines, at first embedded in the colourless sheath but afterwards, by the gradual disappearance of the latter, quite free. An inner, colourless wall, the endospore, bounds the protoplasm of the spore. At maturity, the gelatinous remains of the spore-bearing hyphae entirely disappear and the fully formed spores, which become uninucleate by fusion of the two nuclei, lie quite free in dense masses.

The mature spore is round or elliptical, 7 to 12 $\mu$  in diameter, and with a clear brown wall, furnished with small, rather scattered spines. The Indian specimens do not germinate readily in boiled water when fresh (less than 1 per cent.) and in Europe they are said to require a resting period of 8 to 12 months before they will germinate in water. In the United States, however, they apparently germinate freely in water, as soon as ripe. In nutrient solutions, such as a decoction of manure, immediate germination can readily be obtained. A 4 or 5-celled promycelium is formed, which bears spindle-shaped sporidia at the tip and laterally near the septa. In water, these sporidia sometimes bud off secondary sporidia while still attached, this being especially the case when the promycelium extends out of the water into the air. In nutrient solutions, this budding is much more vigorous, long branching chains of sporidia arising from the promycelium and continuing to bud off new ones even after they become detached. These chains frequently project into the air above the level of the liquid and are, therefore, suited for air-dissemination. Sometimes the promycelium or the earlier formed sporidia, immersed in the liquid, grow into a hypha which reaches the surface and there forms a chain of aerial sporidia. Very often the whole promycelium is thrown off and continues to bud out sporidia after separating from the spore. The sporidia themselves may increase in size in the liquid and even become septate before budding off new sporidia.

In artificial cultures of the budding sporidia, large, dark brown, round bodies, resembling the spores found in the host plant (except that the wall is smooth),<sup>2</sup> have been observed, under certain conditions. They are formed either directly from the sporidia by septation and rounding off of the component cells, or from the germ-tubes which similarly segment. They are believed to represent an early stage of true spore formation, and if this view is correct, furnish one of the very few cases known in which true smut spores have been obtained in artificial culture under saprophytic conditions.

Few smuts are known which grow so vigorously as saprophytes as this species, and the saprophytic life may be prolonged almost indefinitely by transferring at intervals to fresh nutrient solution. At any time, under suitable conditions and especially when in contact with the surface of a maize plant, the sporidium can develop a germ-tube instead of a new sporidium, and this is capable of penetrating the young tissues and forming a smut boil. The sporidia have been shown to remain alive on a glass slip for 5 months and also to stand freezing. The spores themselves are much more resistant and will even retain their germinative capacity after passing through the digestive canal of animals fed with smutted plants. As the sporidia continue to develop vigorously in cattle manure, it can readily be understood why the use of such manure has often been observed to lead to a smutted crop. The production of aerial chains of sporidia, so characteristic of this species, enables them to be readily blown on to all parts of the maize plant. Infection appears to result only from sporidia, or the hyphae which arise from them, not directly from the primary spores; and the chances of the parasite reaching susceptible parts of the host plant would be relatively small, were it not for the copious production of these aerial chains of sporidia.

In maize grown for fodder, smut does not cause any appreciable damage. The plants are not in any way hindered in their growth by the localised infection, as a rule, and are even said to be preferred by cattle. The chief losses occur where the cobs are severely affected in maize grown for grain.

Seed treatment is clearly of no use in checking this disease, since infection is not often from the seed. The only real remedy is to remove, as soon as they appear, all smut tumours (or the whole plant in severe cases) and burn the infective material. Since organic matter favours the development of the sporidia where spores have lodged in it, care should be taken not to expose manure heaps and the like to contamination from spores or diseased plants. For the same reason, the use of cattle manure is not advisable where maize smut is bad. Rotation is important, as the spores may remain in the soil and germinate the following year. The seed may occasionally carry spores, so it is advisable to select it from healthy fields.

The only other plant known to be attacked by this fungus is the fodder grass *Euchlaena mexicana*, which, as already mentioned, is also liable to maize rust and is closely related to maize.

**Head smut (*Ustilago Reiliana* Kuehn).**—This smut is readily distinguished from *Ustilago Zeæ*, as it does not form tumours on the host plant and is confined to the male and female inflorescences; the stem escapes attack, while the upper leaves rarely bear a few sori. It is known in the United States, southern Europe, Africa, Australia, and Japan. In some of these countries (as Australia) it is commoner than the ordinary smut, but usually it is comparatively rare. In India, it occurs chiefly in Kashmir where it is probably more destructive than *Ustilago Zeæ*, and sporadically in the Punjab and northern Bombay. Elsewhere, though found on jowar, it has not been seen on maize. It was first described from Egyptian specimens collected in 1868 and has been known from India, under the name *Ustilago pulveracea*, Cke., since 1876.

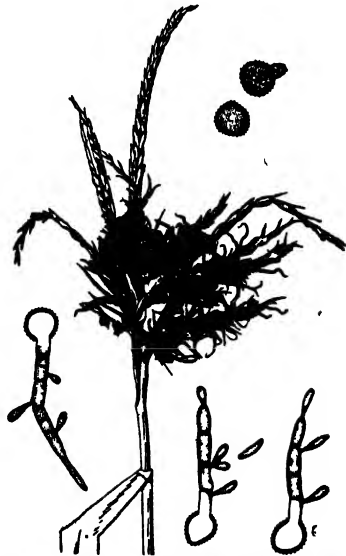


FIG. 69. Head smut of maize (*Ustilago Reiliana*): smutted tassel,  $\times \frac{1}{2}$ ; spores and their germination,  $\times 450$ .

Head smut is usually first noticed on the tassel, sometimes converting the whole upper part to a mass of smut, sometimes affecting single flowers or a portion of the inflorescence only. It forms a rather hard, rough, black mass, not soft tumours like *Ustilago Zeæ*. In some cases, sori are not produced, but the affected flowers grow out into leafy structures and even into small shoots. When sori form in individual flowers, the latter are often much enlarged and deformed. Usually when the tassel shows the smut, all the cobs below are destroyed, though occasionally one may escape or be only partially attacked. On the other hand, the tassel may escape when the cobs are all involved. In the cob, the sorus usually takes the form of a single large smut-mass, enclosed in the bracts and surrounded when young with a smooth or lobed membrane, which encloses the whole part that should have borne grain. The rudimentary cobs often found in the lower parts of the plant may not become smut sori but be transformed into twisted leafy masses. Side shoots may be similarly deformed. Rarely, a few sori are found on the upper leaves or on the bracts around the cob.

The smutted plants are often stunted in growth, brittle, and remain green longer than the rest of the crop. These characters help in detecting the disease, which is often less easily seen than common smut as the sori may remain completely hidden by the enveloping bracts. It is not uncommon to find both kinds of smut on the same plant.

The sori are variable in size, from a quarter of an inch or less on single flowers of the tassel, to several inches long on the cob. They are enclosed in a delicate, pinkish-white membrane, which ruptures at maturity, exposing the black spore-mass. In large sori, the fibro-vascular tissues of the host persist as a stringy, fibrous mass, surrounded by the spores.

The spores are round or somewhat angular, of a rather opaque brown colour, and measure 10 to 16 $\mu$  in diameter, the surface being furnished with numerous minute papillæ. They are darker brown than in *Ustilago Zeæ* and the spines smaller and more densely arranged. Mingled with them are groups of hyaline, sterile cells, sub-spherical in shape and 7 to 15 $\mu$  in diameter. The enclosing membrane is formed of similar cells. When young, the spores are aggregated into masses, but at maturity they separate from one another readily when disturbed. Hence the species is intermediate in its characters between the genera *Ustilago* and *Sporosporium*, and has been placed in the latter by several writers.

Germination occurs readily in water with the production of a thick, straight promycelium, which is usually 4-celled and not uncommonly branched. Sporidia are borne terminally and near the septa; they may sprout into a few secondary sporidia but their development in water is feeble. In nutrient solutions, however, a very copious production of sporidia occurs, branching of the promycelium is common, and chains of secondary sporidia, from which long hyphæ may grow, are formed. The spores have been germinated after being kept for about eight years, and even the sporidia can survive for several months, if kept dry. They germinate best at temperatures between 80° and 90°F. but survive even severe freezing.

The method of infection has been chiefly worked out on jowar, but doubtless follows the same course in maize. Flower infection, such as occurs in the loose smuts of wheat and barley, does not take place in this smut, nor is the infection mainly due to spores carried on the seed coats, as in oat smut and bunt. The main infection comes from the soil and takes place usually during the early stage of seedling growth. But the period during which successful infection occurs is not sharply limited to a definite stage of the seedling, as in oat smut, for it may take place even after the first leaf has emerged and begun to turn green, and successful inoculations have been reported on seedlings about 5 inches high. It is possible also that even later infections may occur, through the lateral buds at the nodes, as there is some evidence that the disease may spread from plant to plant when healthy plants are growing in the neighbourhood of those bearing smut sori. The finer details of infection and growth in the tissues have not been followed out.

The disease resembles flag smut of wheat in infecting from the soil, and seed disinfection has been found to be even less satisfactory than with that smut in checking infection. Formalin, copper sulphate, and hot water have all been tested, with unsatisfactory results.

The spores are shed from diseased plants on the soil around them and remain alive till the following season. Hence smutted plants should be removed as soon as they can be detected. Rotation is evidently of considerable importance, but further work is required to determine the range of dissemination of the spores and the length of time they can remain alive when exposed to seasonal varieties of soil moisture and temperature.

The parasite also attacks jowar.

**Leaf blight** (*Helminthosporium turcicum* Pass.).—This is a fairly common disease in many parts of India and is the only maize disease that has been observed every year to a greater or less extent at Pusa. It was first described in Italy in 1876, and has since been reported in the United States, South Africa, Japan, the Philippine Islands and indeed in most maize-growing countries.

The attack begins as small, yellowish, round or oval spots on the leaves. These extend along the leaf, often limited for a time by the veins, and coalesce into longitudinal bands, which may cover a great part of the leaf. The affected tissues become thin and semi-transparent; then they gradually darken and are covered, in moist weather, with velvety, dark greenish patches, due to the fructifications of the fungus. The attacked leaves gradually dry up and have a burnt or frost-bitten appearance. In severe attacks, the plants may be stunted and the ears poorly developed; sometimes they are said to be killed, but the usual effect is only to check growth and weaken vitality, so that the stalks and grain fail to mature properly.

The fungus resembles the allied species which attack barley and oats, but has not been as fully studied. The conidiophores arise in clusters from the stomata on the surface of the diseased spots. They are long, narrow, unbranched hyphae, slightly bent or curved above, septate, yellowish-brown, and up to  $150\mu$  long by  $6\mu$  broad. The conidia are borne singly at the tips and fall off readily. They are long, rather broad bodies, with 3 to 7 transverse septa, somewhat narrowed at the ends and constricted at the septa, greenish-yellow to brown in colour, and 80 to 120 by 20 to  $24\mu$  in diameter.

The conidia germinate by a germ-tube from one or both ends, and in culture new spores appear in a few weeks. They readily infect young healthy maize leaves, causing spots to appear in about a week.

Serious damage has been recorded from this parasite in Italy and the

United States. In the Philippines also, a bad attack was reported a few years ago, on imported maize varieties, when the crop was about  $4\frac{1}{2}$  feet high. The local varieties escaped damage. In India, the growth is sometimes checked and there is a good deal of loss of leaf in individual plants, but the total injury seems to be slight. In South Africa, where it threatens to be a serious disease, the variety known as Natal White Horsetooth is said to be almost immune.



FIG. 70. Maize leaf blight (*Helminthosporium turcicum*): top of affected shoot,  $\times \frac{1}{2}$ ; conidiophores, conidia, and germination,  $\times 170$ .

No treatment is known. It is probable that the fungus persists on plant debris in the soil, and rotation is therefore advisable. It is suggested in the United States that it may be carried by manure from animals fed on diseased leaves. It is not known if seed infection occurs as in the allied diseases of barley.

The fungus causes a similar disease in jowar.



**JOWAR OR SORGHUM** (*Andropogon Sorghum* Brot.)

**Downy mildew** (*Sclerospora graminicola* (Sacc) Schroet. var. *Andropogonis-Sorghu* Kulk.) — This disease is prevalent in Bombay and is also found in Madras, but has not yet been seen in other parts of India or in any other country. The same parasite (or a variety of it) also attacks several other plants, and as it has been most fully studied on bajra (*Pennisetum typhordeum*), a more complete description will be found under that crop.

On jowar, three distinct types of attack have been described. In the



FIG. 71. Downy mildew of jowar (*Sclerospora graminicola* var. *Andropogonis Sorghu*) affected seedling.

first form, the disease appears in the seedlings just after they come up (Fig. 71). Affected plants have pale yellow, narrow leaves, covered with a fine white down consisting of the conidial stage of the fungus. This

occurs on both surfaces, but chiefly underneath. The plants continue to grow and, when about 5 or 6 weeks old, white streaks appear on the upper leaves. The tissues then tear along these streaks, causing a well-marked shredding of the leaf, which may result in the separation of all the leaf tissue from the midrib in long strips. At the same time, the white areas turn brown from the production of quantities of oospores immersed in the leaf, and similar oospores may appear in the upper sheaths. The plants remain stunted and sterile, no ears being formed. Only individual plants are attacked and there is no indication of spread from plant to plant. This form of attack is exceedingly like that which occurs on hajra, except for the peculiar effects on the ear of the latter.



FIG. 72. Downy mildew of jowar : healthy and diseased ears.

In the second form, the first symptoms are noticed when the plants are about two months old. The top leaves turn white, as also do the bases of the lower leaves. Irregular brown or yellow streaks then appear and oospores develop in quantity in these. On the lower leaves, pale yellow

patches appear, on which the conidial form is found. This may occur at the same time as, or even later than the production of oospores. Affected plants rarely produce ears or if they do the ears are small and with only a few small grains, but they are not malformed as in bajra (Fig. 72). This is the commonest and most prominent form and seems to do most damage, but there is no indication of spread from plant to plant.

In the third form, the affected plants are in groups and the disease seems to spread from one plant to another. The leaves are marked by long, narrow streaks and patches, at first pale yellow, then orange, and finally dark brown. Conidia are found on both surfaces, especially on the under side, but no oospores are produced. The patches appear to spread from the lower to the upper leaves and from the apex to the base of individual leaves. They are limited by the main veins. When the attack is severe, the whole leaf dries up and turns dark brown, but there is no leaf-shredding. Normal ears are produced.

In Madras, it has been noticed that the disease is generally (though not always) most severe in the wetter parts of fields. In some forms of attack the internodes are shortened and the leaves arise close together and stand out stiffly from the stem for some distance before curving over. Hence the Tamils call the disease by a name suggesting the resemblance of affected plants to the screw-pine (*Pandanus*). It has also been observed that the young shoot-buds, which sometimes develop laterally on the stalk, may be attacked and become deformed, remaining imprisoned in the sheath of the subtending leaf.

The microscopic characters of the parasite agree with those more fully described below under bajra, except in certain particulars. The conidia are roundish instead of oval (though with approximately the same limits of measurement as on bajra) and they germinate invariably by a germ-tube and not by zoospores. The ultimate branches of the sporophore are also longer, being up to  $10\mu$  as against about  $8\mu$  in the bajra fungus. It is not improbable that these differences will eventually be considered of sufficient importance to constitute distinct species, but the life-history and, in particular, the ability of these fungi to pass from one host to another, is not yet sufficiently known for a decision to be arrived at. Attempts at cross-inoculation have hitherto failed, but have been few in number.

The disease is not at present a serious one, but it belongs to a group which has dangerous possibilities. No treatment is known, but probably the steady eradication of all affected plants would gradually bring about its disappearance.

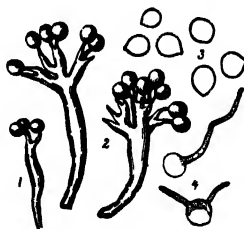


FIG. 73. *Sclerospora graminicola* var. *Andropogonis-Sorghum*: 1, conidiophore with young conidia; 2, older stages; 3, conidia; 4, germination.

**Rust (*Puccinia purpurea* Cke.).**—This is one of the commonest rusts of cultivated crops in India, though the statement formerly made that it is found on most of the millets and wild grasses in the Plains is devoid of foundation. It has only been found here on *Andropogon halepensis* (which many consider to be the wild form from which sorghum developed) though it is reported to attack maize in South Africa. It is known in the Mediterranean basin, south, east and west Africa, the United States, the West Indies, Australia, and throughout southern and eastern Asia. It is not known whether it has an aecidial stage on some other plant, like several allied species.

The parasite causes the appearance of bright purplish-red spots

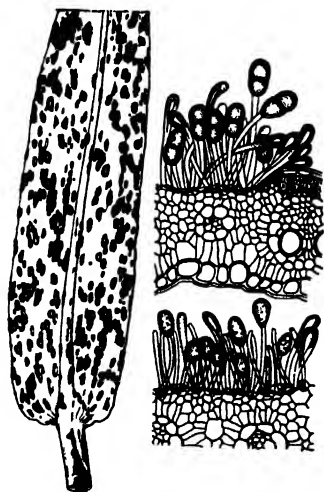


FIG. 74. Jowar rust (*Puccinia purpurea*).  
leaf with spots,  $\times \frac{1}{2}$ ; spore details (after  
Buse),  $\times 100$ .

which sometimes cover a considerable proportion of the leaf surface. In these spots, especially on the under side of the leaf, small, brown or reddish uredo pustules, elongated, parallel to the veins and always situated between the latter, appear. The pustules are scattered or in small irregular groups; sometimes two or more unite. At first they are covered by the epidermis, but this soon ruptures, exposing the light brown spore-powder.

Later on, other similar sori of a deep reddish-brown are found in the same spots, chiefly on the under surface. These are the teleuto sori. As in *Puccinia graminis*, teleutospores may also develop in the uredo sori, causing the colour of the latter to

deepen. When separate, the teleuto sori remain long covered by the epidermis.

The uredospores are ovate or elliptical, with the base flattened where it joins the stalk, and measure 30 to 40 by 20 to 30 $\mu$  in diameter. The epispore is yellowish-brown and furnished with low, almost colourless, tubercular spines, and there are 4 or 5 germ-pores about the middle. Mingled with the uredospores are numerous, large, irregularly club-shaped paraphyses, which vary in colour from pale yellow to brown or reddish-brown, the deepest tint being usually at the tip, where the wall is often much thickened.

The teleutospores are deep brown, generally elliptical, with both ends rounded, slightly constricted at the septum, and with a thick, smooth epispore, sometimes broader at the apex. They are 40 to 50 by 22 to 30 $\mu$  in diameter and are borne on long, persistent stalks, up to 100 $\mu$  in length. Paraphyses, quite similar to those in the uredo sori, also accompany the teleutospores. The latter germinate readily in water after a few days and give rise to typical promyoelia, bearing colourless, oval sporidia, 15 by 10 $\mu$  in diameter, on long sterigmata. These sporidia are not known to be able to infect any plant.

The extent of the injury caused by jowar rust is doubtful. On the one hand, statements have been made that it ranks amongst the most destructive diseases in India, the annual yield of grain being greatly diminished as a result of the attack. On the other hand, it has been noticed in Madras to be most abundant when the crop is ripening and to affect the lower and older leaves, which have already for the most part done their work. It is probable that the injury caused by it has been exaggerated. No doubt it is common to find "light" ears of jowar, but rust does not seem to be the chief cause of this condition. Some, perhaps the majority of cases, are due to the attacks of other fungi or insects, such as the downy mildew already described, stem borers, plant lice, and plant bugs. Others are probably due to unfavourable climatic or other conditions and the attacks of the cereal mould (*Cladosporium*) described above on p. 177.

In the southern parts of India, rust and other jowar diseases depending on air-borne infection are much favoured by the practice of sowing successive crops of this cereal throughout most of the year; there is probably no period when the fresh uredospores are not being liberated into the air. In Bombay also, the fungus has been found on both monsoon and cold-weather jowar. On the other hand, in those parts of northern India where only a single crop is raised, the uredospores have less chance of remaining alive until the following crop is ripe for infection and it is an observed fact that the rust is less common in these localities. In Bihar, for instance, it has been rarely seen, and here the uredospores would have (if infection is conveyed by them) to remain alive in the soil or on decomposing straw through the cold weather and some of the hot weather months, before the new crop would be ready to take infection. It must not be forgotten, however, that the fungus is able to live on the wild variety, *Andropogon halepensis*, and this grass (known as "Baru") persists longer into the cold weather than the cultivated form.

There is no accurate knowledge of the relative susceptibility to rust of different varieties of jowar, though it has been stated that some races are resistant. Should it be found that the disease is more serious than it is at present believed to be, this point would have great importance.

As already explained under wheat rust, practically the only method of checking this class of disease is by the introduction into cultivation of rust-resisting varieties of the host plant. Should these varieties be of inferior quality in other respects, it may be still possible, by the application of modern methods of plant breeding, to improve them up to the required standard. But, so far as is known at present, there is no immediate need for active measures of this nature.

*Puccinia purpurea* has been reported also on bajra (*Pennisetum typhoideum*) in India, but this seems to have been due to a mistake in the identity of either the fungus or the host plant. A second rust of jowar, with smaller uredospores, is said to have been found in Madras some years ago, but has not again been seen.

**Grain smut** (*Sphaceolotheca Sorghi* (Lk.) Clinton).—This is the commonest smut of jowar in India, as in most countries where the crop is cultivated. It is probably, too, the most destructive disease due to a smut in this country, causing great losses in Madras, the Central Provinces, Bombay, and Burma. Even in north India it is prevalent in some places, as in Dehra Dun, but it is very rarely seen in the neighbourhood of Pusa, where, however, the crop is of little importance.

Individual grains only are attacked. As a rule, the majority of the grains in an affected ear are involved, each being transformed into a spore-sac which varies in shape and size according, apparently, to the variety of jowar concerned. Generally the grain is replaced by an oval or cylindrical, dirty-grey sac, sometimes conical at the tip, and measuring from  $\frac{1}{8}$  to  $\frac{1}{2}$  inch in length by  $\frac{1}{12}$  to  $\frac{1}{8}$  inch or more in breadth. This is surrounded at the base by the unaltered glumes. Sometimes the stamens develop normally (Fig. 75, 4), but more usually they are wanting or are involved in the sorus, being represented by three conical protrusions from the sides of the spore-sac (Fig. 75, 5). The stigma is ordinarily not included in the sorus. Spores are not formed in any other part of the plant. In certain varieties of jowar, the shape and size of infected grains are not altered and no elongated sac is produced, but the grain, when broken, is found to be filled with smut powder. In these hidden forms, the disease may easily escape detection until about harvest time, when some of the grains are often ruptured and expose their black powder. They can also often be recognised with practice by the reddish colour of the envelope of the affected grains. Sometimes the ear is only partially smutted, the remaining grain developing normally. Affected plants are not altered in any other way than by the changes in individual grains.

The wall of the spore-sac varies according to the character of the attack. In the long, protruding sacs, the wall is almost entirely composed of fungus tissue in the form of a parenchyma of small cells, 9 to  $13\mu$  in diameter. Only at the base is the wall composed partly of the tissues of the host plant. These sacs rupture and shed their spores more easily than in the next type. In the hidden forms, the wall is in great part the ordinary wall of the ripe ovary, which is tough and rigid and remains unbroken usually until after harvest.

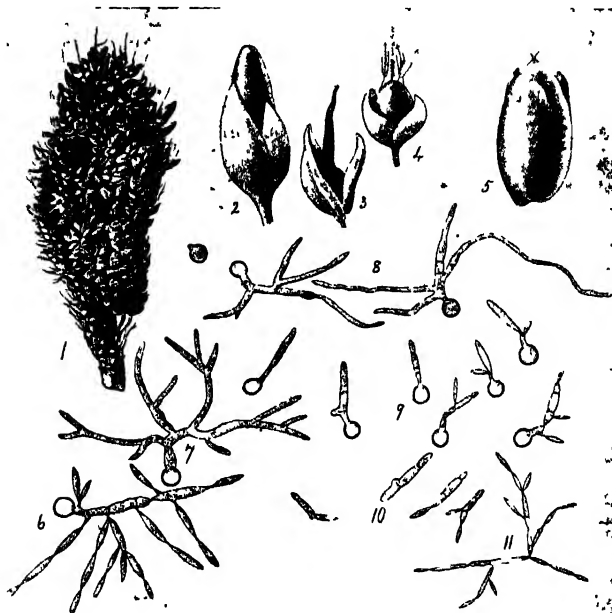


FIG. 75. Grain smut of jowar (*Sphacelotheca Sorghi*): 1, smutted ear,  $\times \frac{1}{2}$ ; 2, a sorus magnified; 3, columella left after the spores have fallen; 4, sorus not involving stamens; 5, sorus with stamens involved; 6, spore germinating with promycelium and sporidia,  $\times 450$ ; 7, spore germinating with branched germ-tube,  $\times 450$ ; 8, promycelia with hyphae in place of sporidia,  $\times 450$ ; 9, promycelia, some constricted at first septum preparatory to being cast off,  $\times 450$ ; 10, cast promycelia,  $\times 450$ ; 11, promycelium with budding chains of sporidia,  $\times 450$ .

The interior of the sac is entirely filled with the spore-powder, excepting a slender central column of hard tissue, which is hollowed into depressions at the surface, filled with spores (Fig. 75, 3). This "columella" is composed of the tissues of the host plant, consisting of parenchyma traversed by fibro-vascular bundles. Occasionally it is branched above, the spore-sacs being sometimes lobed to correspond with the branches. These lobes represent the transformed stamens.

The spores are round to shortly oval, dark brown in mass but brownish-olive singly, smooth, and 5 to 9 $\mu$  (generally about 6 $\mu$ ) in diameter. They are often united into loose masses, which break up into the individual spores as soon as placed in water.

Germination may take place immediately, or after the spores have been kept dry for a long period—up to at least 8 $\frac{1}{2}$  years it is said. They have been germinated in India after two years. In water there are two distinct types of germination, either of which may predominate in any individual specimen. In one type, a typical promycelium of 4 cells is formed, which buds off sporidia laterally and from the apex (Fig. 75, 6). The sporidia are spindle-shaped, 10 to 12.5 by 2 to 3 $\mu$  in diameter, and do not bud off secondary sporidia readily. Clamp-connections and buckle-joints occur, as in oat smut. In the other form, the promycelium is not distinct and sporidia are not usually formed, but a branching germ-tube arises directly from the spore (Fig. 75, 7). Intermediate types may be found, in which a 4-celled structure similar to a promycelium is produced, but, instead of sporidia, long, branching hyphae arise terminally and laterally (Fig. 75, 8). In nutrient solutions, germination is much more vigorous and a promycelium (or sometimes two) with sporidia is generally formed. This promycelium is often markedly constricted at the top of the basal segment and may be sharply bent at this point. Sporidia may be borne at this stage, but more usually the constriction deepens until the whole upper part of the promycelium is abjoined from the spore and thrown free in the liquid, before any sporidia are formed and even before the upper two septa of the promycelium can be seen without special staining methods (Fig. 75, 9-10). On both ends of the fallen part of the promycelium and also laterally near the two septa (which are now distinct) sporidia then appear. These sporidia are longer than those formed in water culture, being often up to 20 $\mu$  in length, and sometimes become divided by one or more transverse septa and increase in size after becoming detached from the parent stalk. They bud out long, branching chains of secondary sporidia, similar to themselves (Fig. 75, 11). Sporidia are also sometimes borne on the tip of the basal segment left still attached to the spore. The formation of a branching germ-tube, or the production of hyphae instead of sporidia on the promycelium, is less often observed in nutrient solution than in water, but has been seen in several cases; as the nutrition becomes exhausted, the sporidia also send out fine hyphae instead of budding off new sporidia.

The life-history of this smut is similar to that of bunt or oat smut. Infection can only occur between the time of germination of the seed and the appearance of the seedling above ground. This time varies according to temperature and moisture. Jowar germinates best between 36° and 40°C., the rate decreasing progressively down to 10°C. The primary shoot grows more quickly the more the soil moisture, up to at least 20 per cent. by weight of water. Infection occurs best on slow germinating seed checked by cold, since the optimum temperature for germination of the spores is only from 20° to 30°C. The fine hyphae which arise from the promycelium, as well as those given out by the sporidia, can penetrate the young tissues of the shoot and give a mycelium in the growing point. Entry seems to be chiefly through the mesocotyl. No outward effect is produced until the ear begins to form. Then the hyphae accumulate in the immature ovary below the epidermis, forming a solid mass of considerable depth. The outer part of this remains unaltered to form the membrane of the spore-sac, which may either protrude well above the grain or remain buried in the ovary. The spores are formed in the rest of the fungal mass, progressively from without inward. The membrane is tough and may persist until after harvest.

The losses caused are sometimes very heavy. From 20 to 25 per cent. of smutted ears have sometimes been seen in India, and the value of the grain destroyed is estimated in Bombay to exceed a million sterling annually and for India as a whole must be several millions.



The treatment is based on the same considerations as in oat smut. Since infection can only occur at the time of germination of the seed, and comes for the most part from spores adhering to the surface of the grain at the time of sowing, seed disinfection is successful, when properly carried out, in preventing all but a very small proportion of cases. Any of the ordinary methods of seed disinfection described under the other cereal smuts can be used. Very good results have been obtained with formalin, and as the grain is particularly resistant to the action of anti-septics, it is safe to immerse it in  $\frac{1}{2}$  per cent. solution for two hours, drying rapidly afterwards. Copper sulphate is also very satisfactory and is widely used in India, this being probably the only smut against which any measures are regularly taken by Indian cultivators. From  $\frac{1}{2}$  to 3 per cent. may be used without appreciably affecting germination. A 2 per cent. solution is employed in the Central Provinces, being sprinkled on the grain, which is well turned over and then spread out to dry. In Bombay and Madras, the grain is immersed in a solution of the same strength for 10 to 15 minutes. The hot water treatment has been advocated in other countries, though more troublesome than the above methods and without any superiority over them, so far as is known. The grain is first heated in warm water for a few minutes and then immersed in water at about 135°F. for 10 to 15 minutes. Below 130° does not kill the spores and above 140° is dangerous to the grain. In all these methods it is advisable to remove unbroken spore-sacs, which, being lighter than the sound grain, will float in water and can be skimmed off. These unbroken spore-masses are said to resist a considerably higher temperature than 135°F. for 15 minutes and are also not easily killed by fungicides. On the whole, the formalin treatment is to be preferred theoretically in this as in the allied smuts, but the copper sulphate method has become so well known that the material is available in many villages and this is a practical advantage. In Bombay, for instance, there is a large demand for one-anna packets, which contain enough copper sulphate to treat 4 acres. It is estimated that some 200,000 acres were sown with treated seed in 1915. There is field evidence that the spores do not persist in the soil from a previous crop (probably because they germinate during wet weather) and effective seed disinfection by copper sulphate or formalin can practically eliminate the disease.

The fungus attacks all the wild and cultivated varieties of *Andropogon Sorghum* (including *A. halepensis*) except the kind known as Milo, which is said in the United States to be practically immune to this and the following smuts.

**Loose smut** (*Sphacelotheca cruenta* (Kuehn) Potter).—This species is frequently confused with the last, the differences being such as readily escape detection. It is common in parts of Bombay and occurs also in the Central Provinces and doubtless elsewhere, being confined to this host, if we except the allied wild species *Andropogon halepensis*. In Bombay, it occurs chiefly on the winter (rabi) crop, especially in the Sholapur district, but is also found to some extent on the rains (kharif) crop. It is known also in Europe, the United States, and Africa.

Affected plants may sometimes be detected before the ears appear, being somewhat stunted in growth, with thinner culms than normal, and with a tendency to tiller. The smutted ears may come out earlier than



FIG. 76. Loose smut of jowar (*Sphacelotheca cruenta*): 1, smutted ear,  $\times \frac{1}{2}$ ; 2, sorus involving stamens, magnified; 3, columella left after the spores have fallen; 4, spores and their germination,  $\times 450$ .

in the rest of the crop, occasionally very much so. The ears themselves are somewhat looser than in unaffected plants; all the spikelets are usually smutted, though occasionally some escape or are transformed into leafy structures; and the glumes are much hypertrophied and may

reach a length of over an inch. Hence this smut is more easily seen than the last, the loose, powdery, black sori which replace the grain being often very prominent.

The sori develop in the essential organs of the flower, the stamens and pistil being transformed into a conical body, or the latter forms an oblong, swollen mass crowned at the top by the stigma and surrounded by the stamens; these may remain free or be each transformed into a sorus united with the pistillar sorus at the base (Fig. 76, 2). The sori measure from 3 to 18 by 2 to 4 mm., and are surrounded by a thin, grey membrane; this is much more transient than in the grain smut and usually ruptures, exposing the spores, before the head emerges from its sheath. In the centre of each sorus, a solid, narrowly conical body, pointed at the tip and extending nearly the whole length of the sorus, is formed (Fig. 76, 3). This is a columella similar to that described in the grain smut, but longer and more curved. When the stamens are involved, each may have its own columella, united at the base with that of the pistil. Sori are also found commonly on the rachis and its branches and sometimes on the glumes.

The spores are formed as in the grain smut, in irregular or elongated clumps, becoming separate as they mature. They are round or shortly elliptical, darker brown than in *Sphacelotheca Sorghi*, with an epispore marked by indistinct pittings, and 5 to 10 $\mu$  in diameter. The membrane is composed of roundish fungal cells, which separate easily into groups and may be found mingled with the spores, as small clumps of hyaline cells about twice the diameter of the spores.

Germination occurs as readily as in grain smut, with the production of a thick, usually 4-celled promycelium, which buds off sporidia sparsely in water, but copiously in nutrient solutions. The sporidia have been maintained in artificial cultures for several months and in some cases (in Africa) the cultures have been carried on to the formation of spore-like bodies. Instead of sporidia, branched hyphae may develop from the promycelium or the older sporidia. Fusions between the sporidia have been observed.

The life-history of this fungus is similar to that of the grain smut. Infection takes place in the seedling stage from spores that have reached the grain coats during harvest and are therefore sown with the seed. Soil infection seems to be rare, presumably because of the readiness with which the spores germinate as soon as the moisture conditions are favourable. The sporidial stage in this group of fungi is known to be usually less resistant to drought and other unfavourable conditions than the spores themselves, and once germination has occurred the fungus finds it difficult to maintain its vitality until the next crop is sown. Though exact evidence is wanting, experience suggests that something of the sort must occur in this smut as, unlike *Ustilago Roiliana*, once the spores adhering to the grain are killed, infection is rarely found.

Hence the seed treatment recommended against grain smut is effective against this smut also. This has been the experience in India and is also true for the United States.

**Head smut (*Ustilago Reiliana* Kuehn).**—As already stated, head smut is found on jowar as well as maize. It is, indeed, much commoner on the former host in some parts of India, notably Bombay where it occurs sporadically everywhere, while head smut of maize is only found in the northern parts of the Presidency and is rare even there. Else-

where this smut has been found in the Punjab, United Provinces, Central Provinces, and Madras, but is not known as yet in eastern India. It has been recorded in the United States, southern Europe, Africa, and eastern Asia.

As in maize, the whole plant is usually infected and a large sorus is normally found in place of each ear. Sometimes the terminal panicle may escape when those lower down are smutted, but in these cases the unsmutted ear remains sterile. Occasionally, partial infection of an ear may occur, the smut-free part being also sterile in this case.

The attacked plants are not noticeably different in size and appearance from the healthy, until the large

FIG. 77. Head smut of jowar (*Ustilago Reiliana*): smutted ears,  $\times \frac{1}{2}$ ; spores and their germination,  $\times 300$ .

sori develop. The latter may be up to 4 inches long by 2 inches broad, being amongst the largest known, and resemble those caused by the parasite on maize cobs. It is not easy to find unbroken sori, as the



membrane is transient and usually ruptures before the bud has emerged from its sheathing leaves, exposing the black spore-masses. Smaller sori may occur on the leaf blades, especially those surrounding smutted ears towards the top of the plant. The membrane is composed of fungal cells, and inside it is a dense mass of spores, traversed by many solid filaments inextricably tangled into a network, between the meshes of which the spores are held. At harvest time only this network may remain, the bulk of the spores having been dispersed by the wind.

The spore characters and germination have already been described under maize. The spores average about  $13\mu$  in diameter and have not proved easy to germinate in Bombay, though the same fungus from maize in Kashmir germinated readily. Artificial cultures of the sporidia have been obtained in the United States and continued growth by budding out fresh conidia for a considerable period, some of the cultures even progressing as far as the formation of spore-like bodies. The life-history, so far as it is known, has already been referred to. Cases of double infection by this species and by *Sphacelotheca Sorghi* and *S. cruenta* respectively have been recorded.

Treatment by seed disinfection, which is so widely practised in the Central Provinces, Bombay, and Madras, fails to prevent this smut. This is because the spores shed on the soil in large numbers remain alive and capable of germination until the next crop is sown. In the two smuts of this crop already described, the spores germinate very readily, and those that reach the soil probably germinate for the most part as soon as the first rain falls after harvest. The sporidia thus produced are unable to survive until the next crop is put in. Hence unless fresh spores are carried in on the grain coats during sowing, the crop escapes attack and the disease may be almost entirely eliminated by disinfecting the seed before sowing. In *Ustilago Rediana*, however, it has been observed that there is a difficulty in getting the living spores from jowar to germinate freely. A few may germinate each time rain falls but there will be many still alive when the next crop is sown. Those that happen to germinate in contact with the young seedling and are thus in a position to cause infection will naturally not be numerous and this probably explains the curiously sporadic nature of the attack.

In the few fields where treatment is required, all attacked plants should be early removed before they shed their spores, and jowar should not be sown again for a few years. An immune variety, Milo, is known in the United States, and resistant kinds may be found in India should the disease increase so as to become dangerous. The young sorus is sometimes eaten by children in Bombay.

As with the last two smuts, this species also attacks *Andropogon halepensis*.

**Long smut** (*Tolyposporium filiferum* Busse).—This is one of the less common smuts of jowar in India, having so far been found in the Madras Presidency and Sind only. It was first described from East Africa but is not known elsewhere.

Like the grain smut, only individual grains are transformed into smut sori, but these are few in number in each ear, not, as in grain smut,

covering the whole or a greater part of the ear (Fig. 78). Each is surrounded by perfectly normal grain and, being very prominent and often widely scattered, are much more readily seen than in *Sphacelotheca Sorghi*. The sorus develops in the ovary as in the latter smut, but is much larger (Fig. 79, 1). The spore-sac is a light brownish-yellow body, from  $\frac{3}{4}$  to 1 inch in length by  $\frac{1}{4}$  to  $\frac{1}{2}$  inch broad. It is often curved and early ruptures at the tip, exposing the spores and also a bundle of 8 or 10 dark brown filaments, the isolated fibro-vascular bundles of the hypertrophied ovary.

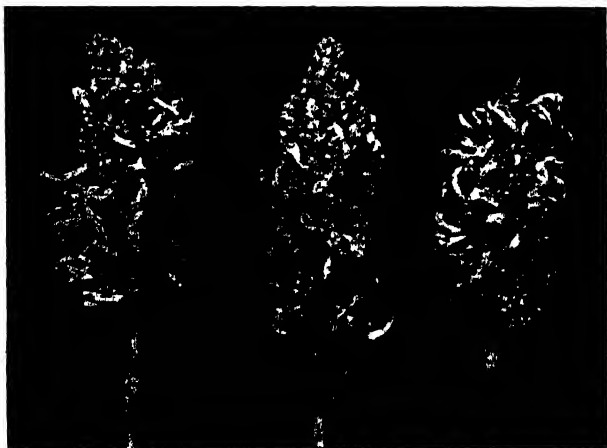


FIG. 78. Long smut of jowar (*Tolyposporium fliferum*).

The spores are held in a rather solid mass amongst the filaments and between these and the wall. They are united into groups, composed of a large number of spores which do not easily separate from one another even when placed in water. This formation of persistent spore-balls is a characteristic of the genus *Tolyposporium*. The individual spores are round, ovate, or flattened, brownish-green in colour, and 12 to 16 by 11 to 16  $\mu$  in diameter. The episporium is densely covered with flattened warts on the free side.

The spores germinate readily in nutrient solutions, all the spores of a cluster being fertile. They have been germinated when nearly two years old. The promycelium is usually 3-celled and bears sporidia at the tip and laterally from near the septa, often in great numbers. These sporidia may bud off others or grow out into long germ-tubes, which may again bear sporidia. At the surface of the liquid, branching chains of aerial sporidia are formed. Often, instead of a true promycelium, the spore germinates by giving out a thick hypha, the protoplasm of which collects in the upper growing part, the emptied part below being cut off by successively-formed septa; sporidia may be produced near the tip or on lateral branches of this hypha.

The life-history of the fungus is quite unknown. The development of the sori in individual, often widely scattered grains of the ear, tells against a generalised infection, such as occurs in grain smut. Possibly each sorus arises from a localised infection of a single flower; the formation of aerial sporidia, similar to those found in *Ustilago Zeæ*, supports this view.

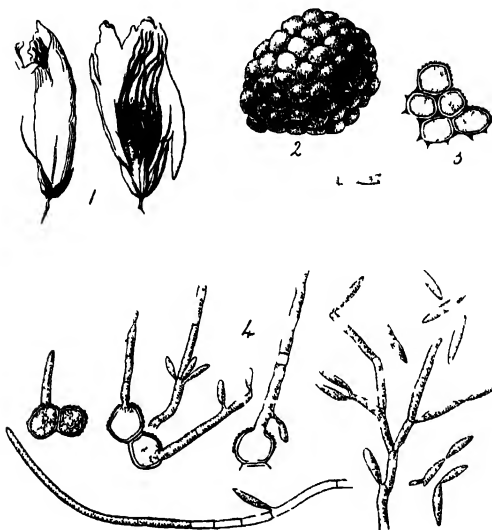
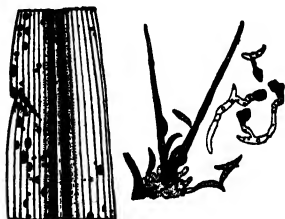


FIG 79 Long smut of jowar (*Tolyposporium fliferum*): 1, ruptured ear; 2, a spore ball; 3, part of same in section,  $\times 450$ ; 4, germination,  $\times 450$ .

No recommendations for treatment can be given, except to destroy the infected ears and obtain seed from healthy fields. The disease is not serious at present and special treatment is probably unnecessary.

**Red leaf spot** (*Colletotrichum graminicolum* (Ces.) Wils.=*C. Lineola* Corda).—This fungus is probably the commonest parasite of jowar in India, commoner even than the rust, and is apparently found wherever the crop is cultivated, having received various names in different countries. Occasionally it has been reported to have damaged the crop in India, but usually the effects are slight, as each spot is small and strictly localised, the rest of the leaf being uninjured and able to perform its functions in a normal manner.

The disease appears as small red spots, visible on both surfaces of the leaf and measuring up to 4 mm. in the longest diameter, parallel to the axis of the leaf. The mature spot is lighter in the centre, where one or more sharp black dots can be seen on either surface. These are the fructifications of the fungus.



. 80. Red leaf spot of jowar (*Colletotrichum graminicolum*): part of leaf with spots,  $\times \frac{1}{2}$ ; part of a sorus with setae and conidia, and germination of the latter,  $\times 170$ .

The mycelium is strictly localised to the red spots, sending hyaline, septate hyphae into the leaf cells of the whole thickness of the leaf, and killing them. Small masses of stromatic tissue are formed in the epidermis and in the air spaces below the stomata on both surfaces. From the outer layers of these stromata, numerous short, hyaline spore-stalks arise, and by their growth the epidermis is ruptured and the outer part of the stroma becomes visible. Each stalk (basidium) then produces a single conidium

at its tip. The conidia are hyaline, falcate, with a single or sometimes two oil drops in the centre, and measure 20 to 30 by 4.5 to 5  $\mu$  in diameter. Other cells of the stroma grow out into long, rigid, black bristles (setae), characteristic of the genus. These are up to 175  $\mu$  long by 4 to 6  $\mu$  broad, sooty-black, unbranched, and with several septa. They taper towards the tip, which is lighter in colour and rounded or occasionally mucronate.

The fungus, though common in many parts of the world, does not seem to have been cultivated and its life-history is unknown. Treatment is impracticable, but fortunately the disease rarely does harm.

The same species is found also on several other grasses, but it is not known if it can pass from one host to another.

**Leaf Blight** (*Helminthosporium turcicum* Pass.).—This fungus, or a very similar form, also attacks jowar in India, Egypt, China and elsewhere. It is not very common or injurious on this host. It has been described above under maize (p. 201).

### BAJRA (*Pennisetum typhoideum* Rich.).

**Green ear disease or downy mildew** (*Sclerospora graminicola* (Sacc.) Schroet.).—This curious disease occurs sporadically in many parts of India, usually resulting in the complete loss of the grain in affected plants. It is also known in East Africa on the same host, while it is found on jowar, *Andropogon halepensis*, *Setaria italica*, and *Euchlana mexicana* in India, and on various species of *Setaria* in Europe, the United States, and Japan. It is not usually of sufficient intensity to attract much notice,





FIG. 81. Downy mildew of bajra (*Scierospora graminicola*) affected ears

but at times, particularly in low-lying, ill-drained land, it develops so as to cause considerable damage.

As the name indicates, the chief symptom of the disease is the transformation of the solid spikeate ear wholly or in part into a loose, green head, composed of a mass of small, twisted leaves. In some cases only a small part of the ear is affected the rest ripening its grain normally, usually, however, complete or nearly complete sterility results.

This is due generally to the conversion of the upper segment of the floral axis, which in perfect flowers develops into the grain into a small, leafy shoot (Fig 33, 1). The other organs of the flower are also altered: the bristles of the spikelets become hypertrophied, sometimes reaching an inch in length and being variously contorted, the fertile glumes enlarged and sometimes turned green, and the stamens much altered or suppressed. Sometimes two spikelets arise from the one pedicel instead of the usual one, and the number of florets on a spikelet may be increased from two to three or four. The main changes are those involving the essential parts of the flower: the stamens and pistil. The former are often converted into minute leaf-like bodies, with a distinct division into leaf sheath and leaf blade (Fig 33, 2) or sometimes are represented merely by brown pointed structures with no trace of the anther. The relative sizes of filament and anther may be profoundly altered and pollen may be entirely absent. The pistil is never developed, being replaced in most cases by the small, leafy shoot referred to above, but sometimes by a minute branched shoot with undeveloped buds, or a simple horn-like growth which appears to be a leaf united along its edges. Naturally, as a result of these changes, the appearance of the ear is greatly modified and diseased plants can be very easily picked out.

Besides the ears the leaves of plants affected with this disease show minor changes. In young plants, in the early stage of the disease, many leaves can be seen with the usual fresh green colour changed wholly or in part to whitish, and later to brown. The whitening of young leaves is visible usually as long streaks, often occupying half of the breadth of the leaf and extending throughout almost its whole length. In older plants, the leaves affected are chiefly those enclosing the ear, which are more completely whitened than in younger plants and more rapidly turn brown afterwards. These leaves are often deformed, being twisted and crinkled, and tend to split into shreds at the tip. In bajra, this shredding is not usually so pronounced as in jowar or *Setaria italica*. The shoot buds which sometimes arise laterally on the stalk are deformed in the same manner as in jowar (Fig 82).

These symptoms are caused by a parasitic fungus which is found chiefly in the leaves of the host plant. The earliest stage of its development which has been detected is in the leaves of young plants under a foot in height. Following on the appearance of the whitish streaks, which are the first outward sign of the presence of the parasite, a cloud of thick sporangioophores bursts from the stomata, covering the surface of the streaks (chiefly the under surface) with a greyish-white, downy growth. The sporangioophores arise from an internal mycelium, which is collected chiefly between the cells of the mesophyll bordering on the fibrovascular bundles and also in the tissues between the bundles. The hyphae grow mainly between the cells but send haustoria into them (Fig 16). Some

hyphae also cross the cells in the manner described above under maize smut, being isolated from the cell contents by a cellulose sheath formed around them by the protoplasm as a protective measure (Fig. 37, 5). The bundles are never penetrated by the parasite (Fig. 37, 6) and the epidermal layer is usually also free, though rarely hyphae occur in the internal wall and send haustoria into the cell cavity. The effect of the parasite on the host plant is slight in the early stages. There is sometimes a little thickening of the leaf due to multiplication of the leaf cells. The bundles are increased in size by a multiplication of both xylem and phloem elements. The whitening of the leaves is due to gradual disappearance of chlorophyll from the assimilating cells, and starch is also noticeably absent from cells containing the haustoria of the fungus. Sporangium-formation is accompanied by great assimilatory activity on the part of the fungus, leading to the collapse and death of the mesophyll cells. As a result of this collapse, the bundles are often flattened or otherwise distorted in the older leaves.



FIG. 82. Downy mildew of bajra (*Sclerospora graminicola*): distorted leaf bud; sporangiophore and sporangia,  $\times 400$ ; latter germinating,  $\times 500$ ; an oogonium with oospore,  $\times 550$ .

When the sporangial stage is about to appear, a small clump of hyphae develops in the air space below each stoma, and from this, one or more sporangiophores emerge through the opening to the surface of the leaf. The sporangiophores are broad, shortish stalks, measuring about  $100\mu$  in length, by  $12$  to  $15\mu$  broad, unbranched in the lower part, but usually with a few short, thick branches, dichotomously or trichotomously

formed, at the top, and crowned by numerous papillæ of characteristic shape, on which the sporangia are borne. The latter are hyaline, broadly elliptical, slightly pointed at the free end, with a thin, smooth wall, and measure 19 to 31 by 12 to 21 $\mu$  in diameter. They fall early without any stalk, and germinate rapidly in water, liberating from 3 to 12 or more zoospores, of an irregular kidney-shape, furnished with two cilia from the concave side. After swimming for a time, the zoospores come to rest, round off, (measuring 9 to 12 $\mu$  when quiescent), and germinate rapidly by a hypha. The sporangial stage has only been found on young plants not yet in ear, and appears to be often altogether absent. After the fall of the sporangia, the sporangiophores soon collapse, the whole superficial growth of the parasite being evanescent and easily overlooked.

At a later stage in the life-history of the parasite, oospores are produced in immense numbers. They can generally be found in any plant bearing the green ears characteristic of the disease, but do not appear to develop in young plants. They are formed in organs which have turned brown as a result of the attack, being developed chiefly in the mesophyll of the leaf blades, both of the foliage leaves and of those formed in the inflorescence. Very rarely they occur in the sheaths of the foliage leaves and in the glumes, while in contrast to the foliage leaves, the sheaths of inflorescence leaves often contain them. They are large, reddish-brown bodies, just visible to the naked eye, especially when viewed by transmitted light. As oogonium-formation takes place chiefly in the parenchyma bounding the bundles, rows of the brown dots may often be seen on each side of the leaf veins. This position of greatest development of the fungus is the cause of the tendency to shredding of the leaves, mentioned above, the tissues between the veins being in great part destroyed. The ripe oogonium, after fertilisation, thickens its wall, which is closely applied but not united to the wall of the oospore. The whole fruit is characterised by the possession of a very thick wall in two layers, the outer of which is the oogonial wall, and only the inner belongs to the oospore. In spite of numerous attempts, these spores have not been germinated in India nor, so far as is known, elsewhere.

The oospore is usually quite spherical, with a smooth, yellow wall of even thickness all round. It measures 22.5 to 35 $\mu$  in diameter, averaging 32 $\mu$ . The oogonial wall is tawny in colour, irregularly thickened, and the whole fruit, as a result of these thickenings, is not round but angular, elliptical, or irregular in shape. It measures 34 to 52 $\mu$  in diameter, averaging 42 $\mu$ .

The life-history of the parasite is not known. The sporangial stage is evanescent or often absent and the sporangia themselves are short-lived bodies, so that this stage cannot be of importance in propagating the disease from year to year. Indeed the sporangia appear to be functionless, as there is no indication of spread from plant to plant and attempts to inoculate healthy leaves with the zoospores have failed. It is probable that the disease is carried over by the oospores, which are admirably suited for the purpose owing to the very resistant wall. It appears regularly in certain places every year and may be caused by germination of the oospores after a resting period in the soil, though all attempts to germinate them artificially have failed. Another alternative is that the sound grains produced not infrequently on diseased plants become infected by growth of the internal mycelium into them from below, and subsequently give rise to diseased plants. To test this a considerable number of such grains, carefully collected from partially diseased ears, were sown, but all gave healthy plants. Microscopic

examination has also failed to reveal the mycelium in such grains, and the disease has not been checked by soaking the grain for 5 minutes in hot water at 65°C., a temperature which few internal parasites could withstand.

The question whether the fungus can pass from one to another of its hosts is also undecided. Experiments carried out in Italy seem to show that *Sclerospora graminicola* cannot pass from *Setaria italica* to *Setaria verticillata* or *Setaria viridis*, two other known hosts for the fungus, while in India the bajra fungus will not attack jowar leaves and *vice versa*. It is also noticeable that the fungus in some localities in India is confined to one host, even though some of the others may be growing near by. Thus in Pusa it has only been found on *Setaria italica*, though jowar and bajra have been grown in the vicinity. This suggests that the parasite has split into specialised races, each confined to one or more hosts, as in the cereal rusts.

The only recommendation for treatment that can be given in the present state of our knowledge is to pick out and destroy infected plants, so as to prevent the oospores being disseminated.

It may be noted that of the other hosts mentioned as susceptible to the attacks of this fungus, both sporangial and oosporeal stages have been found on all but *Euchlæna mexicana*, on which the former alone has been found. In this case there is therefore some doubt as to the identity of the parasite, since members of this group are not easily distinguished by the sporangial stage alone, and the nearly-related maize parasite, *Sclerospora Maydis* (p. 191), may be the species here concerned. An allied fungus, *Sclerospora macrospora*, attacks wheat, oats, maize, and a number of wild grasses in South Europe and has also been reported on rice in Japan. In this species only the sexual stage is known, sporangia having never been observed.

**Rust** (*Puccinia Penniseti* Zimm.).—This fungus is found in Madras, Bombay, the Central Provinces, the United Provinces, and Bihar. Outside India it has only been reported from East Africa.

It forms small, brownish-yellow pustules in groups, arranged often in longitudinal series, on the leaves and stem. Later on, as the second spore form appears, the pustules become blackish. The leaf may turn brown around the sori, especially when young, but old leaves are not extensively discoloured.

The uredo sori are found on both surfaces and are very variable in size and shape. In severe attacks they become confluent and much of the epidermis is lifted up in flakes

around them. The uredospores are oval or pear shaped, yellow, 33 to 38 by 23 to 30 $\mu$  in diameter, the wall with scattered spines and four equatorial germ pores, and the stalk

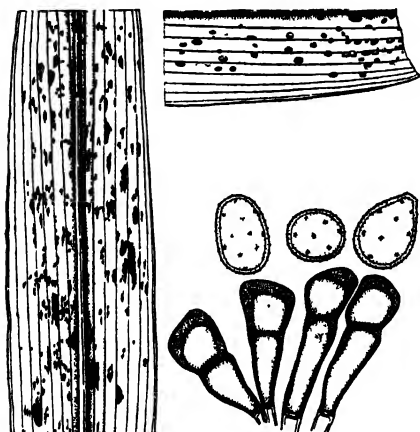


FIG. 83. Bajra rust (*Puccinia Penniseti*): part of leaves with uredo (on left) and teleuto (on right, above) sori,  $\times \frac{1}{2}$ ; spore details,  $\times 390$ .

colourless and shorter than the spore. The teleuto sori resemble those of the uredo except that they are brown in colour. The teleutospores are bright brown, cylindrical to long oval, rounded or flattened at the tip, which has a much thickened wall, slightly narrowed at the septum, and smooth. They measure 40 to 60 by 16 to 20 $\mu$  and have a short, colourless stalk. The apical germ-pore is indistinct, but they germinate at the apex in the usual way, with the production of a 4-celled promycelium which bears elliptical sporidia, 11 by 6 $\mu$  in diameter. Paraphyses are not found in either spore form.

The asexual host, if one exist, is unknown and the fungus has doubtless dispensed with this stage in its normal life, as in so

many allied species

No remedial measures are practicable, but fortunately the damage appears to be slight as a rule.

**Smut (*Tolyposporium Penicillariæ* Bref.).**—The smut of bajra has hitherto been found only in parts of Bombay and Madras, and at Pusa in a crop grown from Bombay seed. Outside India, it has not been definitely identified but appears to occur in Egypt and possibly in other parts of Africa.

As in the allied *Tolyposporium filiferum* on jowar, the attack is visible only in scattered grains in the ear, the majority of the grains developing normally. Sometimes the affected grains are single, sometimes grouped in patches of varying size, frequently confined to one side or towards the base of the ear.

The sori occur in the ovary as oval or pear-shaped bodies. These project clearly beyond the glumes and may be from half to twice the diameter of the normal grains, being often 3 or 4 mm. long by 2 or 3 mm. broad at the top, which is bluntly round to conical in shape. Their

colour varies in a remarkable manner, being sometimes bright green, occasionally chocolate-brown, and often, especially when old, dirty black. The colour is due to the membrane, the included spore-mass being always deep brown to black. The membrane is tough and the spore-mass compact, granular, and not easily dispersed by the wind.

In microscopic structure, the spore-mass is found to be composed of a great number of roundish balls of spores, each ball measuring from 40 to 150 $\mu$  in diameter and consisting of a large number of spores, tightly pressed together so as to break up with difficulty even when placed in water. The individual spores are round or angular, 8 to 12.5 $\mu$  in diameter, and with a light brown, irregular or slightly roughened wall. The membrane of the sorus is composed of the tissues of the host, lined inside with fungous elements. There is no columella.

Germination is scanty and difficult to obtain by the usual methods. The promycelia are formed from the spores while still attached in masses, and radiate out around the spore-balls. The promycelium is normally 4-celled and forms sporidia terminally and laterally. It is peculiar in having a tendency to break up into its individual cells, which fall off and continue to bud out sporidia until nutrition begins to fail, when they may grow into hyphae. When such hyphae emerge from the liquid in which they are formed, they again give off branching chains of sporidia. These arial conidia may be formed in such numbers as to cause a definite layer on the surface of the liquid.

The life-history is not further known. Artificial infections have not been tried, but at Pusa the disease increased in intensity in the plots in which bajra was grown, for some years after its first appearance. All attempts to check the smut by seed treatment failed and the same has been the experience in Bombay. Formalin, copper sulphate, and hot water have all been tried without success. Even immersion for 10 minutes in water at 60°C. or for 5 minutes at 65°C. failed, though the last treatment reduced the germination of the seed by 50 per cent.

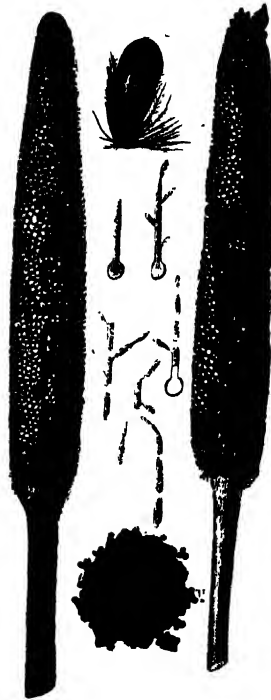


FIG. 84. Bajra smut (*Polyporus Penicillaria*): affected ears,  $\times \frac{1}{2}$ ; above, a single sorus,  $\times 3\frac{1}{2}$ ; below, a spore-ball; in centre, germination of the spores,  $\times 300$ .

From European experiments, which showed that fresh spores received from India would not germinate and that success was first obtained by keeping them dry for a year and then for a second year buried in moist earth, it would seem that the species is particularly well equipped for preserving its vitality, when the spores reach the soil, until the next crop is sown. This, coupled with the failure to check the disease by seed disinfection, suggests that the attack usually comes from the soil, as in flag smut of wheat and head smut of maize and jowar. Hence the same measures should be adopted as against these smuts, diseased ears being removed and the crop rotated so as to leave an interval of, say, three years between two successive crops on the same field. The percentage of loss is not ordinarily high and it is doubtful whether it will always pay to remove smutted plants, as they bear a fair amount of grain except in severe attacks.

The fungus is not known on any other plant.

### RICE (*Oryza sativa* L.).

**Bunt** (*Tilletia horrida* Tak.).—This disease occurs throughout the main rice-growing areas in Burma, and has also been found on the east coast of peninsular India and at Pusa. As it is not easily detected in the field, the actual distribution in India may be much more extensive. It was first described in Japan and has since been found to be widely distributed in south and east Asia, including Java, Indo-China, Siam, and China. It has also reached the United States, probably with imported Japanese seed, and became established in South Carolina for a few years, and also in Louisiana.

The spore sori are produced only in a few individual grains in the ear, often not more than two or three on an ear in India, though in the United States as many as 25 per cent. have been noticed. Frequently it is difficult to detect the diseased grains without breaking them, the sorus being almost hidden by the enclosing unaffected glumes. In other cases, the glumes are forced a little apart and a black mass of spores is extruded. The spores are sticky and adhere to the surface of the glumes and even to the outside of healthy grains in contact with the affected ones.

Within the grain, the fungus at first lies outside the aleurone layer, or under the thin membrane which forms the skin of the grain and consists of the wall of the ovary and the outer layers of the true seed. Gradually an extensive cavity is formed between the seed covering and the endosperm; and the spore-mass may entirely replace the starch cells of the seed or may form only a layer of greater or less extent in the endosperm (Fig. 85, 3).



At first it is enclosed in a thin membrane, consisting of the outer skin of the grain, but later on this usually ruptures and the spores lie in a rather compact or adherent mass within the glumes, or protruding slightly from their edges.

The mycelium can be found in the stem of affected plants, running between the cells of the vertical sub-epidermal row of parenchyma, under the stomata. Spores are formed in the ears, beginning as thin-walled terminal swellings on short branches. They are round or rarely elliptical, opaque black when mature, light or dark brown when young. They measure usually from 20 to 24 $\mu$  in diameter. The structure of the walls is peculiar, giving the impression when seen with low powers of the microscope, of a dark band furnished with projecting curved spines. More carefully examined, however, the "spines" are found to be thickened blunt pegs, formed in the substance of a thin, hyaline

membrane, which persists until after maturity. Seen in surface view, the blunt ends of the thickenings appear dark, the spaces between being light, so that a reticular effect of light bands is given (Fig. 85, 5). The hyaline membrane is somewhat gelatinous when moist and the adhesive properties of the spores are due to it. Mixed with the spores are numerous large, yellowish or sub-hyaline cells, which represent, in part, immature spores. Fragments of septate hyphae may also be found between the spores.



FIG. 85. Rice bunt (*Tilletia horrida*): 1, part of bunted ear; 2, a bunted grain; 3, same in cross and longitudinal section; 4, spores,  $\times 215$ ; 5, section and surface view of spore, showing structure of wall,  $\times 900$ .

The spores have only been germinated in Japan and evidently require special conditions for their germination. After 3 days in water they put out a promycelium, septate at the tip, and bearing at its end a cluster of 10 to 20 needle-shaped sporidia, 38 to 53 $\mu$  in length and divided into 3 or 4 cells after falling from the promycelium.

The amount of damage caused by the disease is usually slight, and it is only in South Carolina, where it was vigorously dealt with and died out in some four years after discovery, that it appears to have assumed threatening proportions. In Louisiana, it has only been recorded on Honduras and red rice, though the millers state it occurs occasionally on Japan rice. In India, it is sporadic in its appearance and no preference for particular varieties has as yet been noticed. Attention was first called to it by a statement that its spores were of almost universal occurrence in rice bran imported to Germany from Burma and Siam. As the outer skin of the grain is removed in milling and forms a considerable proportion of the bran, exported chiefly for cattle food; it can readily be

understood that a small proportion of bunted grain with spore-masses formed just below this skin, will contaminate a large sample.

The parasite is allied to those that cause bunt of wheat, and though its life-history has not been worked out, there is reason to believe from the results of the treatment adopted in South Carolina that it enters the young seedling in just the same way, while still below ground. From the characters of the attack, it appears that the whole stool becomes infected.

For prevention, experiments in America showed that it is sufficient to float out the light grains (which include the infected ones) in cold water and then to soak the seed for 24 hours in a solution of  $1\frac{1}{2}$  lb. liver of sulphur in 25 gallons water, or for 2 hours in a 2 per cent. solution. No doubt the ordinary methods used against wheat bunt would be equally successful. No case has as yet been seen in India where treatment would pay, but the members of this group of parasites have a way of multiplying rapidly under favourable circumstances and it is consoling to know that an effective remedy is available in case of need.

The fungus is not known on any other host.

**False smut** (*Ustilaginoidea virens* (Cke.) Tak.).—A disease which resembles in its effects those caused by the smut fungi, but which is really due to an Ascomycete, occurs in all the great rice-growing areas that bound the Bay of Bengal and is also found in the western maritime districts such as Malabar and, sporadically, in inland tracts like Bihar. The parasite was first described from Tinnevely in 1878, as a true smut, under the name *Ustilago virens*, and nine years later was found in Japan and named *Tilletia Oryzæ*. Subsequently it was cultivated artificially in Germany and found to agree with the lower stages of an Ascomycete on *Setaria*, for which the genus *Ustilaginoidea* was founded. The ascleterous stage of the rice parasite has not yet been discovered, but there is no reason to doubt that it will be found to agree with that on *Setaria*. Outside India, it is widely distributed in southern and eastern Asia and is known in Louisiana in the United States.

The parasite is visible only in the ears, where it develops its fructification in the ovary of individual grains. These are transformed into large, velvety, green masses, which may be more than twice the diameter of the normal grain. The green colour is superficial, the inner parts of the swollen mass being orange-yellow near the surface and almost white in the centre. The glumes are unaltered and are found closely applied.



FIG 88. FALSE SMUT OF RICE.



to the centre of the green mass, which bursts out above and laterally from between them; occasionally one of them may be entirely encircled by the fungus. Only a few grains in each ear are usually affected, and these may occupy any part of the ear.

The young ovary is invaded by the parasite at an early stage in its development, and is transformed into a hard mass of closely-united, fine, colourless hyphae. This sclerotium-like body grows and bursts out laterally between the closely-applied glumes. Here its outer layers develop spores of a brownish-green colour when ripe but orange in the mass when immature. As the ripe spores are progressively formed from the surface inwards, the velvety, green appearance of the outer part, changing to orange and then colourless further in, is readily understood. In very old fruits, the colour of the whole sclerotium, except the innermost part, is deep greenish-brown or almost black, the centre being yellowish, and spores being found to a considerable depth.

The centre of the sclerotium is composed of pseudoparenchyma entirely replacing the tissues of the grain. Nearer the surface the hyphae are arranged radially, sparingly branched and septate, while the exterior consists of a mass of loosely adhering spores. The spores are formed laterally or, rarely, terminally on the radial hyphae. The youngest spores are almost colourless and are found on the hyphae bounding the white centre of the sclerotium. Those further out, as well as the hyphae on which they are borne, have a yellowish colour, forming in mass the orange-yellow zone of the sclerotium. Still further out, the mature spores are greenish-brown and the outermost layer consists of these with a few fragments of the sporiferous hyphae, which are ultimately almost entirely used up in the building of the spores. The young spores are smooth or almost so, but when mature they have a rough, olive-green, granular coating, soluble in ammonia, caustic potash, alcohol, and mineral acids. The same substance coats the older hyphae to a lesser degree.

The spores are borne on minute projections from the sides of the radial hyphae, sometimes so close together as almost to hide the latter from view. They are usually round and measure 4 to 6 $\mu$  in diameter, occasionally elongated and 8 by 4 $\mu$ .

They germinate in small numbers in water and nutrient solutions by the formation of slender, septate hyphae, which, after a limited growth, form clusters of minute, pear-shaped, colourless conidia at and near their tips. In the United States, it was found that the spores lose their power of germination after about 6 months. In water, the germ-tube may bear only a single conidium at its tip, but in nutrient solutions the stalk continues to grow from underneath the first-formed conidium, which is pushed over to one side, and a new terminal conidium is formed just above the first. This process may be repeated several times and small heads of conidia formed. The germ-tube may branch, and in Germany and the United States, cultures of branching hyphae, forming a mycelium of

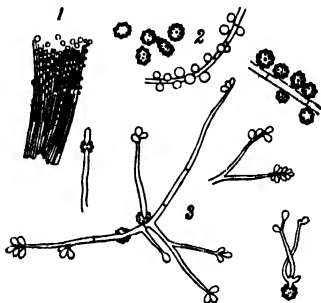


FIG. 87. False smut of rice (*Ustilaginoides virens*): 1, section of outer part of sclerotium; 2, spore-formation and ripe spores,  $\times 450$ ; 3, germination of spores with formation of secondary conidia,  $\times 300$ .

considerable size, have been obtained. In Germany, these have been carried on to the formation of sclerotia, bearing ripe spores similar to those found on affected plants. The conidia will also germinate in nutrient solutions with a short germ-tube, on which still smaller conidia develop.

The further life-history is unknown. There is a yeast-like conidial budding fungus, which develops in cultures and has been taken to be a secondary stage, but this is also readily obtained from healthy plants and is only accidentally associated with Ustilagoidea. It has been named *Cladosporium Chodati*, and plays an important part in the preparation of the hill beers brewed from rice in many parts of India, having considerable fermentative activity. All attempts to reproduce the disease artificially have failed. Numerous efforts have been made, both in India and elsewhere, to cause infection by mixing spores with the seed and also to produce direct infection of the flowers, but without success. Seed from ears bearing diseased grain gives healthy plants. As the spores lose their vitality after some months, infection from the soil is improbable. The scattered sclerotia on an ear and the occurrence of the disease in patches suggest that infection occurs at flowering time and the sclerotia develop rapidly as the grain ripens, but attempts to prove this have failed. No perfect stage has been obtained, in spite of repeated attempts at Pusa and elsewhere.

Hence, in the absence of full knowledge of the life-history, it is not possible to give satisfactory recommendations for treatment. Fortunately the disease is not severe in any part of India, so far as has been ascertained. It is more common than bunt, but still only affects a small percentage of the ears and usually only a few grains on each ear. Even if treatment were known, it would scarcely be worth the trouble of carrying out in most cases seen.

The fungus is not known on any other plant.

**Sclerotial disease** (*Sclerotium Oryzae* Catt.).—A disease of rice caused by this fungus, which is probably responsible in the aggregate for very considerable losses, but which is easily overlooked owing to the obscurity of its symptoms, is widely prevalent in practically all the important rice-growing areas of India. Outside India it is known in Japan and Italy, but has not hitherto attracted much attention.

It is well known that rice is particularly prone to bear "light" ears, that is, ears in which a greater or lesser number of the grains do not mature, the cavity within the glumes remaining empty. The condition is familiar to rice millers and in Burma has received a special name

"gwa-bo." Insect attack, both by borers that penetrate the stem and by sucking insects that damage the flowers, is responsible for much of the trouble; nematode worms do great damage in the same way in certain parts of India; but everywhere cases are found free from animal pests and showing no definite symptoms except that the ears are light and there is a tendency to throw out green shoots from the base when the rest of the crop is ripening and turning yellow.

An examination of mature plants with these green shoots usually shows that the base of the stem is slightly discoloured at the lowest distinct internode or the next one or two above. On splitting the culm, a dark greyish web of hyphæ may be found within the hollow stem, and small, round, black, shining bodies (sclerotia) can be seen dotted all over the inner surface. Sometimes the base of the culm is quite free from the fungus and the attack begins at a node some distance up the stem. The lower leaf sheaths are also often involved and within their rotting tissues rows of sclerotia may form. When one plant in a clump shows the characteristic late shoots at the base, all the other plants in that clump are, as a rule, similarly attacked and every ear has a considerable number, often 50 per cent. or more, of light grains.

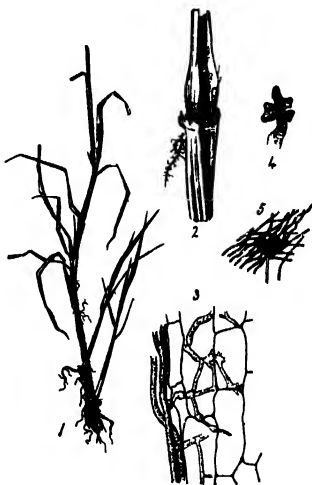


FIG. 88. Sclerotial disease of rice (*Sclerotium Oryzae*). 1, affected plant showing late shoots at base; 2, sclerotia in hollow of culm,  $\times 4$ ; 3, hyphae in air space and cells of leaf,  $\times 200$ ; 4, appendage at end of hypha,  $\times 240$ ; 5, sclerotium from a culture,  $\times 37$ .

The hyphæ of the parasite penetrate the cells and large air spaces found between the main vascular bundles of the culm and leaf sheath. They pass from cell to cell readily, often with a constriction at the point where the wall is penetrated and a swelling of the hypha on the proximal side. They also enter the hollow of the stem and the spaces between the stem and sheaths, forming a grey, woolly growth. Sclerotia are developed on the free mycelium lining the inner surface, and also in the air spaces. The bulk of the hyphæ run longitudinally in the large air cavities and in the cells bordering on them, while a certain number grow outside the tissues between the sheaths. In artificial infections, the infolded leaf blades in the bud are readily penetrated, especially from the inner surface, where the epidermal walls are thin. Young plants thus infected are completely

killed, whereas in the diseased, nearly mature plants seen in the fields, death does not usually follow the attack but only partial sterility and excessive late tillering.

In culture, the mycelium is at first white but later may turn brown, especially at the edges and on the surface of the medium. Peculiar dark-brown appendages may develop at the margin of the growth. These may consist of one or more cells of irregular, often deeply lobed shape, which suggest appressoria. Segmentation of the hyphæ into chlamydospore-like cells, barrel-shaped, with thick, black walls and oily contents, may also occur. Sclerotia begin to form in five or six days, arising from a plexus of interlacing hyphæ, which continue to branch and intertwine until a small, spherical, compact mass is formed. For a time, the young sclerotium increases in size by the adhesion of fresh branches to the periphery. Ultimately, the cell walls turn black and growth ceases. At this stage the interior of the sclerotium has a very definitely parenchymatous structure, and the filamentous character of the hyphæ is lost; along the periphery, however, a thin layer of loosely intertwined, hyaline hyphæ can be seen. The mature sclerotium is roughly circular, smooth, black, and from 150 to 500  $\mu$  in diameter. It shows little differentiation into cortex and medulla, and though it has been said in Italy to become ultimately hollowed out into a cavity containing circular pycnospores, no such change has been observed in Indian specimens and no further stage has been seen.

Artificial inoculations at Pusa have proved this fungus to be a virulent parasite, killing 70 to 80 per cent. of the young plants in about two weeks. Penetration occurs readily into the sound, unwounded tissues. The effect on older plants in the field appears, however, to be rather a gradual weakening of the host, culminating in the failure to produce good seed. The reason for this difference in virulence is not known.

The fungus was first described in Italy, as the cause of extensive damage to the rice crop in certain parts. In Japan also, it has been mentioned as causing much loss of grain. It is, however, exceedingly difficult as yet to apportion the responsibility between the several obscure diseases of rice which result in diminishing the yield, and much further work is required before a satisfactory knowledge of them can be obtained. *Sclerotium Oryzæ* is probably the worst fungus disease of rice as yet identified in India.

Against a parasite such as this, it is difficult to see what remedial measures can be employed with success. The sclerotia of the fungus undoubtedly persist in the soil, where, under favourable conditions, they germinate and produce a mycelium which attacks the crop. The use of soil fungicides is, as has already been pointed out, little likely to be practicable on a field scale in India, and no other method of destroying the sclerotia suggests itself.

### KANGNI OR RALA (*Setaria italica* Beauv.).

**Green ear disease** (*Sclerospora graminicola* Schroet.).—*Sclerospora graminicola* attacks wild and cultivated species of *Setaria* in many parts of the world. In India it causes considerable damage to the Italian millet in Bihar and Madras and has also been collected in Kashmir. The



symptoms of the disease are almost exactly similar to those described at length above, under green ear disease of bajra, but the shredding of the leaves is usually more marked in *Setaria* than in the other cereals attacked by the fungus. In some cases, a considerable part of the grain is lost; when this occurs seed should be obtained from a healthy crop and the land which has borne the diseased crop should not again be sown with *Setaria* for two or three years.

**Rust** (*Uromyces Setariae-italicae* (Diet.) Yosh.).—(Fig. 89, 1—2).—

This rust is not uncommon in India on several species of *Setaria*, including the cultivated varieties of Italian millet. It has been found chiefly in the Peninsula from Poona to Mysore, and is known also in eastern Asia and north Africa, but has not hitherto been reported elsewhere.

Only the uredo stage is usually developed, teleuto-spores having been observed occasionally in Japan and once in Bihar. The uredo sori occur on both surfaces of the leaf, chiefly underneath, as small, oblong, cinnamon-brown pustules, often arranged in rows. The teleuto sori are similar, but remain longer covered by the epidermis and are greyish-black in colour.

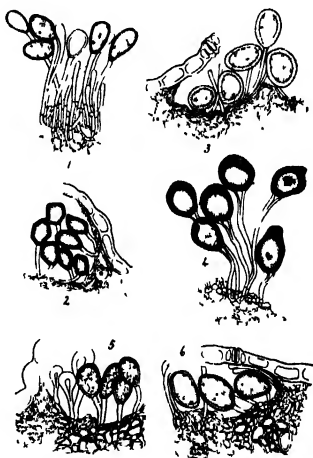


FIG. 80. Rusts of millets: 1—2, *Uromyces Setariae-italicae* on *Setaria italica*,  $\times 190$ ; 3—4, *Uromyces linearis* on *Panicum miliare*,  $\times 235$ ; 5, *Uredo operta* on *Coix Lacryma-Jobi*,  $\times 212$ ; 6, *Uredo Paspali-scorbiculati* on *Paspalum scrobiculatum*,  $\times 190$ .

The uredospores are round or ovate, spiny, yellowish-brown, and 22 to 34 by 18 to 26 $\mu$  in diameter, with 3 or 4 germ-pores. The teleutospores are roundish or oblong, often angular, with a smooth wall, slightly thickened at the apex, and yellowish-brown or yellow in colour. The wall is thicker than that of the uredospores, and the measurement 17 to 30 by 16 to 24 $\mu$ . A hyaline or faintly coloured stalk usually remains attached to the spore when the latter falls off.

It is probable that this rust causes little damage, partly because, as noted in Japan, it appears late, at about the ripening period of the host plant. It has been observed more than once that rusts which attack

the young plant are often the most destructive, as for instance *Puccinia triticea* on wheat.

No treatment has been tried, nor is any likely to be effective, short of breeding resistant varieties.

**Smut (*Ustilago Crameri* Koern.).**—The smut of rala is common in Bombay, especially in the Karnatic, where nearly half of the crop of the

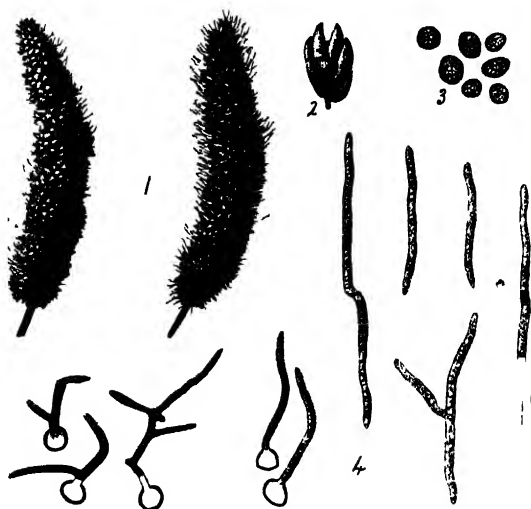


FIG. 90. Smut of *Setaria italica* (*Ustilago Crameri*): 1, healthy and smutted ears,  $\times 1$ ; 2, a smutted grain,  $\times 4$ ; 3, spores,  $\times 450$ ; 4, germination of spores with branched germ-tubes, and tips of latter set free,  $> 450$ .

Presidency is grown. It doubtless occurs in other parts of India but has not yet been recorded. Elsewhere it is known in North America, southern and eastern Europe, China, South Africa, and Japan. In Bombay, it sometimes causes the loss of 20 to 30 per cent. of the crop.

The sori are confined to the flowers, affecting the ovary and basal part of the pales only. As a rule, every shoot is diseased and every flower smutted, though sometimes the upper part of the ear may escape. The normal grains are replaced by somewhat larger, spherical or pear-shaped sori, measuring 2 to 4 mm. in diameter and of a pale greyish-white colour,

which can be readily distinguished from the golden colour of the unaffected ripe ears. Later on, many of the sori rupture and expose the black spore-powder, which is disseminated by the wind. Unruptured sori may, however, be found mixed with the grain even up to sowing time.

The spores are dark brown in mass, lighter singly, of somewhat irregular shape, roundish, oval, or angular, smooth, and 7 to 10  $\mu$  in diameter. They germinate readily in water, producing a long promycelium, divided as a rule into 4 or 5 cells. No sporidia are formed, but in place of them long, slender hyphæ are given off, often from the clamp-connections and buckle-joints developed between two cells. In nutrient solutions, germination follows the same course, but is more vigorous. The lower part of the hyphæ becomes septate and emptied of protoplasm, which collects towards their tips; these may then be set free and grow out into filaments, and new hyphæ of the same type may continue to be given off as long as the nutrient solution remains unexhausted.

Infection takes place in the seedling stage from spores adhering to the grain coat and sown with the seed, just as in oat smut or grain smut of jowar. The histological details have not been worked out, but infection probably follows much the same course as in *Sphacelotheca Sorghi*. There is no evidence that infection from the soil occurs.

Hence treatment is on the same lines as in the grain smut of jowar. Experiments in Bombay showed that the disease can be entirely prevented by steeping for 10 minutes in 2 per cent. copper sulphate, and this method is being introduced into practice. In Europe, the best results have been obtained with formalin. Complete prevention has been obtained by immersing the seed in 0.5 per cent. formalin for 5 minutes, the floating grains being skimmed off and the rest then rinsed with clean water and dried. Almost as good results were got by immersion for three hours in 0.25 per cent. formalin, without removing the floating grains or washing with water. Copper sulphate has not proved so satisfactory in Europe as in India. Only good seed should be used, as old or damaged grain may be injured by the treatment. The grain should be dried quickly or it may begin to sprout, and it should be sown as soon as possible after treatment.

The fungus attacks also the allied grass, *Setaria viridis*,

#### CHENA (*Panicum miliaceum* L.)

**Smut** (*Ustilago Panici-miliacei* (Pers.) Wint.).—This disease resembles the head smut of jowar, the ear being wholly transformed into a large spore-sac, filled with dark spores and the remains of the fibro-vascular bundles of the inflorescence.

Its distribution in India is not known, as it has been found only in Kashmir up to the present time. It also occurs in Japan and south and central Europe on the millet; and in Australia on an allied wild grass.

The smutted ears are long concealed by the sheath of the enclosing leaf, but become visible about harvest time as large conical sacs, the whole



FIG. 91. Smut of *Panicum miliaceum* (*Ustilago Panicis-miliacet*),  $\times 1$ .

ear being replaced by a single sorus, covered by a shining, greyish-white membrane which, as it escapes from the enclosing sheath, early ruptures and exposes the black spore-mass inside. Affected plants remain green even after the healthy ones have turned yellow in ripening, except that the ends of the leaves wither; there is also an excessive development of hairs on the enclosing leaf sheaths. Usually any secondary ears that may form laterally below the main head are equally affected. It is said that in rare cases the whole inflorescence is not destroyed, a few aborted flowers on tortuous peduncles being visible.

The spore-sac is enclosed in a membrane formed of fungus tissue. Inside, the spores lie surrounding numerous fine, hard filaments, which are the fibro-vascular bundles left when the smut tumour forms spores, just as is found in *Ustilago Reiliana*. The spores

develop in a soft parenchyma concentrically around each bundle, the youngest being next the bundle, while older ones are in contact with those formed round neighbouring bundles. Sterile bands of fungus tissue, like that which forms the membrane of the sac, traverse the interior. The spores originate in groups closely united together and surrounded by sterile, gelatinous hyphae which disappear at maturity. These spore-balls break up more

or less readily, when placed in water, and the individual spores are all capable of germination. Owing to the mode of development of the spores in united balls, some authors consider this fungus to belong to the genus *Sorosporium* rather than *Ustilago* and, as in *Ustilago Reiliana*, it is at least intermediate between the two genera.

The spores are roundish, elliptical, or often angular, yellowish-brown in colour, and measure 9 to 13 by 8 to 11 $\mu$  in diameter, the epispore being almost smooth or furnished with very minute punctations. They germinate freely in water, giving rise to a promycelium of just the same type as in *U. Crameri*. Long, branched hyphae, with the contents collected at the ends, are formed in place of sporidia, arising often from the buckle-joints between two cells. In nutrient solutions, however, sporidia are formed in fair numbers, but they soon grow out into hyphae instead of giving colonies of budding spores in the usual way. The hyphae may again form sporidia at their tips, especially where they project into the air. Spores kept dry for 8 years have been found still capable of germination.

The life-history is similar to that of *Ustilago Crameri*, infection taking place in the young seedling soon after germination. Experiments carried out in Japan and Austria have abundantly proved that the smut can be induced by mixing the spores with the seed before sowing. Even with spores two years old, 50 per cent. of the plants in a small plot were infected in this manner in Japan, while a similar plot near by, to which the spores were not added, bore no smut. In some parts of Japan, it is the practice to harvest the ripe healthy ears only. In this way the smut-masses are left behind and, as they do not usually open before harvest, the grain does not get contaminated by smut spores. In these localities the smut does not cause any damage. In others, it is the custom to harvest the entire stalk for thrashing. The smutted ears are broken during the latter process and the spores get disseminated on to the healthy grain. Here the damage caused is great, leading sometimes to the entire loss of a harvest. In Germany, 60 to 100 per cent. of smutted plants have been obtained by spraying the germinated spores on to very young seedlings. There is no evidence that infection occurs from the soil.

The treatment is therefore based on the same considerations as in *U. Crameri*. No experiments have been tried in India, but sufficient proof has been obtained elsewhere that disinfection of the seed is entirely satisfactory in preventing the disease. Copper sulphate and formalin have both been used with success, and may be employed as recommended for *U. Crameri*. As in the latter case, it is important to dry quickly, on account of the rapidity with which the wetted grain germinates; and for the same reason it is sometimes better to use a short formalin treatment ( $\frac{1}{2}$  per cent. for 5 minutes), the floating spore-masses and light grains being skimmed off and the grain immediately dried without washing in water, than the usual  $\frac{1}{2}$  per cent. for 2 or 3 hours

without skimming. Hot water at 55°C. for 7½ to 12 minutes has also been recommended.

The fungus is said to attack also the allied grasses, *Panicum Crusgalli* and *P. spinescens*, in other countries.

**Rust** (*Uredo* sp.).—A rust is found on this crop in India, but occurs only in the *Uredo* stage and has not yet been identified. It would seem to be of little importance, as it has only been collected in the Wynaad.

### KUTKI (*Panicum miliare* Lam.).

**Rust** (*Uromyces linearis* B. and Br.).—(Fig. 89, 3-4).—This fungus is only known in India, Ceylon, and the Philippine Islands, and no information is available as to the extent of the damage which it can cause. It has been reported from near Poona.

The uredo sori appear in linear rows on the upper surface of the leaf and are minute, narrow, brown, and slow in bursting through the epidermis. The spores are round or elliptical, spiny, yellowish brown to brown in colour, and measure 22 to 28 by 20 to 25 $\mu$  in diameter, with a rather thick wall. The teleuto sori are similar to the uredo sori but black. The teleutospores vary from almost round to broad elliptical, the wall being greatly thickened (up to 11 $\mu$ ) at the free end, they are smooth, brown, 20 to 32 by 18 to 25 $\mu$  in diameter, and seated on long, thick stalks which remain attached to the spore when it falls off.

Several of the wild species of *Panicum* are attacked by rusts resembling more or less closely this species, and in India it has been found widely distributed on *P. repens*.

### SAWAN (*Panicum frumentaceum* Roxb.).

**Smut** (*Ustilago Panici-frumentacei* Bref. and *Ustilago paradoxa* Syd. and Butl.).—Two quite distinct smuts are found on this crop.

The first, *U. Panici-frumentacei*, has been collected near Simla, at Pusa, and in the Madras Presidency, and is, therefore, widely distributed in India, though apparently unknown elsewhere.

The sori occur not only in the inflorescence but also on the stem, especially at the nodes, and on young shoots. Both those given off from the ground level and those formed laterally in the axils of the older leaves. These shoot infections cause considerable deformity, a twisted mass of leafy shoots, with sometimes short or aborted ears, and with sori at the nodes or near the apex, being formed. In the ear also, there may be some deformity and twisting of the rachis. The stem sori are large, up to half an inch in diameter, swollen, and covered with a persistent, grey membrane. In the ears, only individual flowers are involved and the majority of

the grains may develop normally. The grain sori are formed in the ovary, which is swollen to twice or three times the normal diameter. On rupture of the membrane, the inside is found to consist of a powdery, black spore-mass.

The spores are round, dark brown, with thick, sharp spines, and 7 to 12 $\mu$  in diameter. They germinate with the formation of a typical promycelium on which, especially in nutrient solutions, a copious production of sporidia takes place, in long, branching chains of a regularly dendritic shape. As the nutrition becomes exhausted, hyphae are produced, which may emerge from the liquid and form small chains of aerial sporidia of the same dendritic type (Fig 93).

The life-history is unknown and no treatment has been tried.

The second smut of sawan, *U. paradoxa*, is equally only known from India, having been observed at Pusa on several occasions. It differs from

the last in producing sori exclusively in the ears and in causing no deformity of the host plant.

The sori are found in individual grains, sometimes only a few on an ear, more often replacing the majority of the grains. They are developed in the ovary, which is transformed into a round, hairy, grey set, up to 2 mm. in diameter and therefore not greatly exceeding the size of the normal grains. The latter can, however, readily be distinguished by their smooth, light coloured, shiny coat. The membrane of the sorus is tough, but when ruptured by rough handling a black, powdery spore-mass is exposed.

The spores are nearly round or oval, smooth, olive-brown, with granular contents, and 7 to 10 $\mu$  in diameter. On germination in water, they give out long, branched, septate hyphae, without any formation of a true promycelium or sporidia.



FIG. 92. Smuts of *Panicum frumentaceum* (*Ustilago Panicis frumentacei* on right and *U. paradoxa* on left),  $\times \frac{1}{2}$ .

As in the other smut of this crop, no treatment can be recommended, since the life-history is entirely unknown.

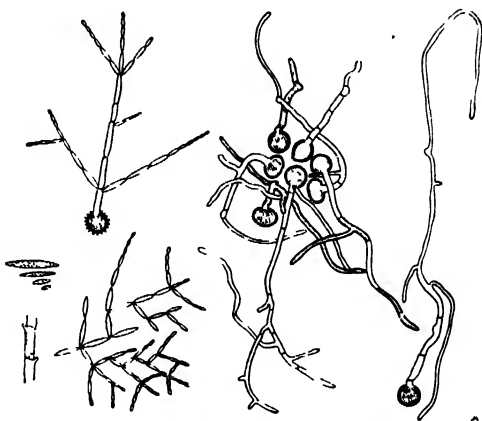


FIG. 93. On left, germination of spore of *Ustilago Panici frumentacei* with formation of sporidia. On right, germination of spores of *U. pasia doza* with formation of branched germ tubes.

### KODRA (*Paspalum scrobiculatum* L.).

**Rust** (*Uredo Paspali-scrobiculati* Syd.).—(Fig. 89. 6).—The rust of this extensively cultivated millet has hitherto only been collected in the Himalaya, eastern India, and Ceylon. It is not believed to be prevalent in the important kodra cultivation of the Central Provinces and Bombay.

The sori are found on the leaves, mostly on the upper surface, as small, scattered, brown, roundish or oblong (up to  $\frac{1}{4}$  mm long) pustules, which remain long covered by the epidermis. The spores are irregularly ellipsoidal, of a bright chestnut-brown, 26 to 40 by 24 to 28  $\mu$  in diameter, and with a spiny wall about 2  $\mu$  thick. A few paraphyses are present in the sori.

The remarks regarding the treatment of other cereal rusts, no doubt apply equally to this species.

**Smut** (*Sorosporium Paspali* McAlp.).—This smut occurs on the cultivated crop in Bihar and is also known in Ceylon and Australia.

It resembles *Ustilago Reiliana* in transforming the whole ear into a smut sorus, which may be 2 or 3 inches long by about  $\frac{1}{2}$  inch broad and is surrounded, when young, by a cream-coloured membrane. The whole is often partly enclosed in the flag-leaf of the ear.



The sori destroy the whole inflorescence except the fibro-vascular bundles of the rachis, which persist as a bundle of filaments.

The spores are formed in loose balls, 30 to 50 $\mu$  in diameter. Individually they are globose-angular or roughly pear-shaped, dark to yellowish-brown, with a thick, smooth wall, and 11 to 18 by 8 to 12 $\mu$  in diameter. They have not been germinated.

Nothing is known of the life-history and treatment has not been tried.

### RAGI OR MANDUA (*Eleusine coracana* Gaertn.).

**Leaf blight** (*Helminthosporium nodulosum* Berk. and Curt.).—The commonest disease of this extensively grown crop in India is caused by a similar fungus to those that cause the leaf blights of barley, maize, and jowar. It has been reported also from the United States and the Philippine Islands.

Affected leaves become marked by numerous elongated spots, at first oval and light brown, then elongating parallel to the axis of the leaf and turning deep brown. The old spots may be up to 1 cm. long by about 1 mm. broad, but as several may unite, larger patches are often formed. The attacked leaves wither prematurely and the grain yield is consequently reduced. The leaf sheaths and even the ears may sometimes become infected.

The fungus forms a small, woolly, greyish-brown mass in the centre of each spot. This consists of clusters of sporophores

arising from an internal mycelium through the stomata and also, though less often, directly across the epidermal cells. The sporophores are erect, unbranched, often much curved, or with prominent knee-bends, deep brown below, paler at the tip, and with numerous septa. They measure from 80 to 250 by 5 to 7 $\mu$  and bear the conidia singly at the tip, the earlier-formed spores being pushed over to the side by renewal of the growth from just below their insertion. The conidia

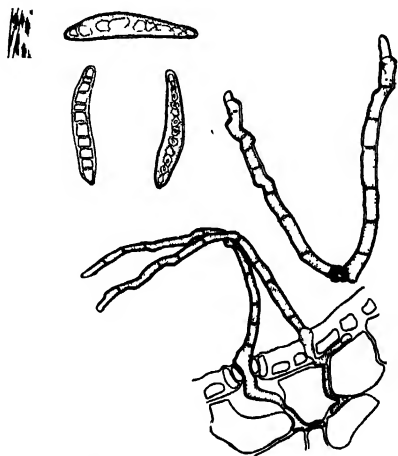


FIG. 94. Leaf blight of *Eleusine coracana* (*Helminthosporium nodulosum*): part of leaf with spots,  $\times \frac{1}{2}$ ; conidiophores and conidia,  $\times 250$ .

fall off very easily and are large bodies, from 50 to 100 by 12 to 17 $\mu$  in diameter, clear brown in colour, straight or slightly curved, and divided by from 4 to 10 transverse septa. Their germination has not been observed.

As the life-history of this fungus is unknown, no suggestions for treatment can be given.

### KASI (*Coix Lachryma-Jobi* L.).

**Rust** (*Uredo operta* Syd. and Butl.).—(Fig. 89, 5).—This inconspicuous fungus attacks the Job's Tears millet in parts of the hill districts of south India and is also known in Ceylon. Usually the leaves are also affected by the black spot due to *Phyllachora Coicis*, and it is not possible to determine the amount of damage caused by these two fungi.

The straw-coloured uredo sori are found scattered on both surfaces of the leaf and are so minute as readily to escape detection. They remain long covered by the epidermis, but eventually rupture to expose the uredospores. The latter are nearly round, elliptical, or egg-shaped, yellowish-brown, and measure 22 to 36 by 18 to 28 $\mu$ . The epispore is thickened and darker at the apex, and provided with spines especially at the upper part. Mixed with the spores are sterile, curved, club-shaped, almost colourless paraphyses.

The disease is probably not susceptible to treatment.

**Smut** (*Ustilago Coicis* Bred.).—The smut of this millet was first found in the Himalaya some twenty years ago, and has since been collected in Bombay and Java. As our knowledge of the cultivation of the crop is still very imperfect, no information is available regarding the amount of damage caused by the disease, but the millet is an important minor crop in some of the hill districts, and since the fungus entirely destroys the grain, it may cause considerable loss.

Every grain of the head is transformed into a black spore-mass, without much increase in size as compared with healthy grains. The spore-mass is surrounded by a membrane hidden by the glumes, and is traversed by flattened or angular filaments, probably the remains of the fibro-vascular bundles of the axis.

The individual spores are dark brown, round or slightly oval, measuring 7 to 9 $\mu$  in diameter, and with a finely spiny epispore. They are mingled with sterile fungus cells and short hyphae. They were germinated in Germany after being kept for two years in damp earth and then sown in nutrient solutions. In Java, they germinated in water and in nutrient media immediately after ripening. The promycelium is always four-celled and, when old spores are germinated, is slightly constricted at the lowest septum, where it tends to break off in much the same way as described above under *Sphacelotheca Sorghi*. Sporidia are formed terminally and laterally near the septa and these may bud off secondary conidia. The primary sporidia also often elongate, after falling, into a separate filament, which may be longer than the promycelium and which also buds off secondary sporidia from the ends and from near the septa.

The life-history of the fungus is unknown and therefore no recommendations for treatment can be given.

**Black leaf spot** (*Phyllachora Coicis* Henn.).—This is caused by a member of a very common group of Ascomycetous leaf parasites, the *Phyllachoras* being found on many grasses, as well as on the leaves of fig and other trees in India. They do not ordinarily cause appreciable damage, being limited to small spots on the leaves, and the surrounding tissues not being in any way affected. The fungus has been found in south India and in various places in southern and eastern Asia.

Affected leaves bear, chiefly on the under surface, numerous shiny black, elongated, flattened stromata, up to 2 mm. long by  $\frac{1}{4}$  to 1 mm. broad, which may be mistaken, on superficial examination, for a rust.

Microscopically, each black body is composed of stromatic fungus tissue, hollowed out into a few flask-shaped or lens-shaped cavities, without distinct walls, and up to  $400\mu$  in longest diameter. These contain asci, cylindrical or clavate in shape, and 50 to 100 by 15 to  $20\mu$  in diameter. The ascospores are eight in number, arranged obliquely in a single row or sometimes double near the top. They are unicellular, elliptical, very pale yellow in colour, and 14 to 22 by 10 to  $14\mu$  in diameter.

As the fungus, though prominent, does little damage, being strictly limited to the blackened spots, no treatment is required.

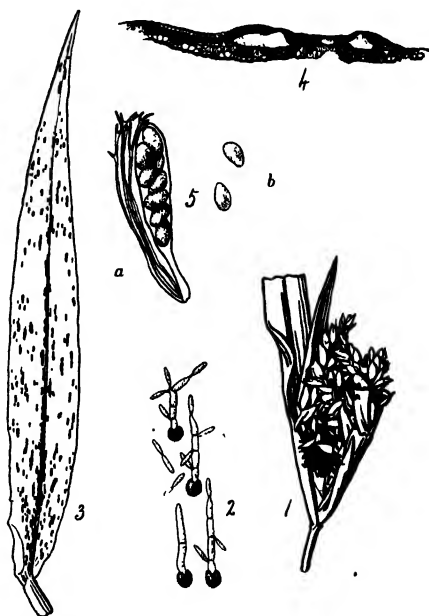


FIG. 85. Smut (*Ustilago Coicis*) and black spot (*Phyllachora Coicis*) of *Coix Lacryma-Jobi*: 1, smutted ear; 2, germination of smut spores with formation of sporidia,  $\times 175$ ; 3, leaf with black spot,  $\times \frac{1}{4}$ ; 4, section of same showing stroma and perithecia; 5, ascus of same, with paraphyses *a* and ascospores *b*,  $\times 250$ . (2 after Brefeld).

## CHAPTER VII.

### PULSE CROPS.

#### PIGEON PEA OR ARHAR (*Cajanus indicus* Spreng.).

**Wilt** (*Fusarium udum* Butl.).—The term "wilt" is applied to a class of diseases very prevalent in India, particularly amongst plants belonging to the pulse and mallow families. It is characterised by the gradual or rather sudden withering and drying up of the green parts, exactly as if they were suffering from drought, even though there may be plenty of water in the soil. Examination of the main roots and of the base of the stem shows that the tissues are blackened, either uniformly or, especially in early stages, in streaks. This blackening is sometimes visible through the bark but is best seen on removing the latter, the wood being the seat of the most pronounced discoloration. In some cases as in the pigeon pea wilt, the black streaks can be traced up the stem to a height of several feet, and the earliest branches to wither are those which arise from the blackened parts of the stem. In all cases, the streaks can be traced down to the roots and are found to arise from main or lateral roots which have become rotten. It is not uncommon to find a plant with one side only withered, with the stem on this side blackened, and with a few of the larger roots on the same side rotten.

These diseases appear to be mostly caused by parasitic fungi belonging to the genus *Fusarium*, which are soil dwellers and are capable of a saprophytic life in the soil for long periods, in the absence of a suitable host plant. They are probably of the class of half-saprophytes, i.e., fungi which are normally saprophytic and find in dead matter (in this case, organic débris in the soil) all the nutriment requisite for their full development. When, however, they encounter the roots of certain plants, they are capable of entering and passing through a period of parasitic life. This behaviour should be contrasted with that of the smut fungi, most of which, while capable of life for a longer or shorter period as saprophytes in soil, manure, and the like, can only reach their full development when growing as parasites in living plants: with one or two doubtful exceptions, the development of the perfect spore stage of smuts only occurs in living plants.



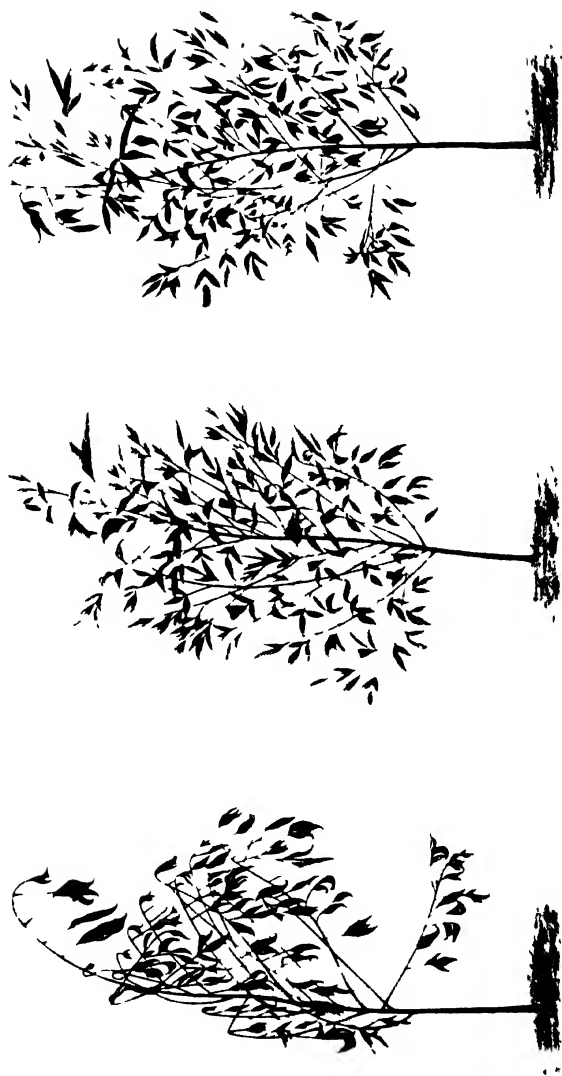


Fig. 96. PIGEON PEA WILT (*Fusarium udum*) middle PLANT HEALTHY, that on right, WILTING; that on left, DEAD.

The genus *Fusarium* belongs to the Deuteromycetes or imperfect fungi, many of which are known to be only stages in the life-history of higher fungi, principally Ascomycetes. *Fusarium* itself is often a conidial stage of Hypocreaceous fungi, chiefly species of *Nectria* and *Neocosmospora*, Ascomycetes possessing bright red perithecia. None of the soil *Fusariums* which cause wilt diseases of plants have, however, as yet been satisfactorily proved to belong to any known Ascomycete; they appear to live from generation to generation in the conidial stage only, and to have lost the ascigerous condition. But there are several non-parasitic species which have retained this stage, and there is a common example in a *Fusarium* on rotten plant roots in soil in many parts of India, which bears under suitable conditions, both on the roots and when grown in artificial culture in the laboratory, the coral-red perithecia of *Neocosmospora vasinfecta*. Even amongst the parasitic species, several which attack the stems of plants and cause cankers commonly develop the *Nectria* type of perithecium at a late stage of their growth. But, so far, the perithecial stage of the forms that attack roots has not been found.

The wilt of pigeon pea is common throughout India, being very destructive in parts of Bombay, the Central Provinces, the United Provinces, and Bihar. It is less frequent in Madras, but has been found as far south as Godavari. In infected parts of Bombay and Bihar, experiments have shown that the loss from the disease may considerably exceed 50 per cent., if the crop is grown on the same soil for two or three years in succession. It does not seem to be the practice, however, in any part of India to grow the crop twice consecutively on the same land, and the care which is exercised in the proper rotation of pigeon pea is not improbably the result of experience of the damage caused by the disease when rotation is neglected. The parasite has not yet been reported outside India.

The symptoms of the disease are, as already stated, such as would arise from a total or partial stoppage of the water-supply from the roots to the aerial parts of the plant. It first appears in the young seedlings a few inches high; at this stage it is usually overlooked unless the number of deaths is large, which is not often the case at such an early period. Usually, if rotation has been properly attended to, only a few isolated plants will be found wilting early in the year. Later on, neighbouring plants, generally those nearest to the early cases, dry up, a month or more often elapsing between the death of one plant and that of its nearest

neighbour. By the time the crop has reached its full height, the deaths will be found to have occurred chiefly in little groups, each corresponding to an early case, and these groups increase in size by centrifugal spread

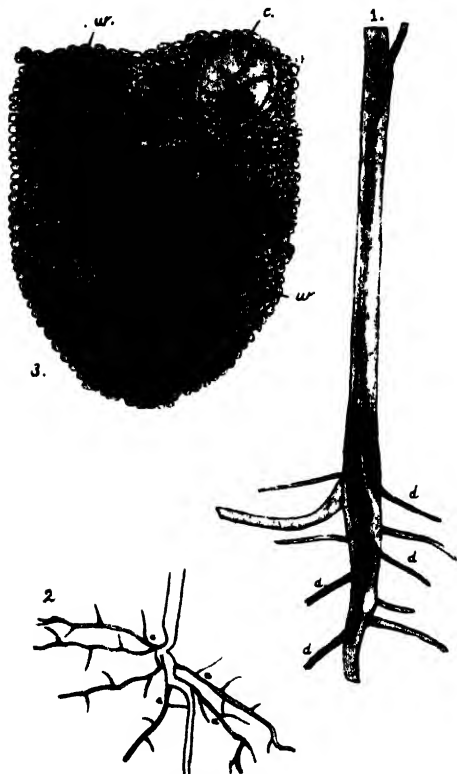


FIG. 97. Pigeon pea wilt (*Fusarium udum*). 1, stem in early stage of attack with bark peeled off to show blackened streaks on the wood, *d* blackened roots; 2, roots of wilted seedling showing points of infection at *a*; 3, hyphae in vessels *w* with microconidia in *c*.

up to the time of harvest. All this time, too, new isolated cases may occur, each serving to originate a fresh wilted patch in the field.

Attacked plants are usually killed. It is not certain, however, that this is always the case, as several instances have been observed in Bihar



of a pigeon pea disease characterised by failure to produce pods, the plants dropping their flowers prematurely or failing altogether to flower. In some of these cases the roots have been found slightly infected by a fungus which appeared to agree with *Fusarium udum*, and hence it is possible that there is a mild form of the disease which causes sterility but does not kill the plants outright.

Microscopic examination of the roots of diseased plants shows that the blackened tissues of affected roots are permeated by the hyphae of a fungus. These accumulate chiefly in the wood, in which the large xylem vessels (whose function it is to carry water, taken up by the roots, to the above-ground parts) are frequently plugged by masses of hyphae, offshoots from which extend into the neighbouring cells (Fig. 97, 5). The hyphae are hyaline and run through the cells, growing with great rapidity along the inside of the walls of the large vessels, up into the stem. This general tendency to rapid longitudinal spread up the xylem explains why the discoloration, which marks the presence of the parasite, may be found at a comparatively early stage in the disease, extending in long streaks up the stem from the point of origin of the infected roots. The preference shown by the fungus for the water channels of its host, and the manner in which these are plugged by matted coils of hyphae, are perhaps sufficient to account for the symptoms of drooping and wilting of the leaves characteristic of the disease. Though the water supply to the roots may be ample, its passage to the green parts is impeded by the choking of the vessels; this may be sufficient to produce the same effect as severe drought; but it is possible that poisonous substances excreted by the fungus may also play an important part, as has been shown to be the case in the bacterial wilt of tobacco (p. 335).

Infection occurs through the fine lateral roots, which are penetrated by the hyphae and rapidly blackened and shrivelled (Fig. 97, 2). The fungus early gains the wood and passes back along this to the larger roots, which are in turn killed. Penetration can occur, in the absence of wounds, through the fine roots, but does not seem to succeed in uninjured large roots or the base of the stem, though it is possible that infection can be carried out here also if the tissues are broken. There is no evidence that it ever takes place above ground.

Spores are produced even within the tissues of the host plant. One form of conidium is borne on short hyphae within the large vessels (Fig. 97, 3 c). This is a small, elliptical or curved spore, formed apically and thrown off by the growth of a second from the same spot. These spores are usually unicellular but may have one or more septa, and measure from 5 to 15 by 2 to 4  $\mu$  in diameter. Within the roots, they usually become free on falling but in artificial culture and on the surface of the plant they are held together in a little drop of liquid at the tip of the hypha, until as many as a dozen have been successively formed (Fig. 98, 3). This type of conidium is characteristic of the imperfect genus *Cephalosporium*, and may be called the *Cephalosporium* or microconidial stage of the fungus. A second kind of spore is also formed within the vessels in old cases: a spherical or oval cell (or often several such united into a short chain) formed from any part of a hypha by the division of the latter into short segments, which round off and become thick-walled. This is a more durable spore than the first type, being capable of germinating after a long interval of time. As it is formed on any part of the mycelium by the simple segmentation of the hypha, it belongs to the class of spore mentioned on p. 14 under the name of chlamydospore (Fig. 98, 8). A third spore form is developed on the surface of the bark on small cushions of stromatic mycelium, which burst out through the epidermis. The spores of this type are long, curved, pointed at the ends, and formed at the tips of short conidiophores as in the first type but falling individually without being held together in a ball. They are divided by from three to five transverse septa and measure 15 to 50 by 3 to 5  $\mu$  in diameter. This is the *Fusarium* or macroconidial stage of the



FIG. 98. *Fusarium udum*. 1-4, microconidia from culture,  $\times 730$ ; 5, formation of same,  $\times 730$ ; 6, germination of same,  $\times 730$ ; 7, macroconidia from culture,  $\times 730$ ; 8, chlamydespores,  $\times 730$ ; 9, germination of macroconidia, some forming secondary spores,  $\times 330$ ; 10, anastomosis of germ tubes of two macroconidia,  $\times 330$ .

fungus (Fig. 98, 7). The other two spore forms are also developed on the surface of the blackened parts; all three may be found together, or encouraged to appear by placing a diseased root or stem in a moist atmosphere for some days. Usually the *Cephalosporium* stage develops first, the *Fusarium* next (but is often wanting), while chlamydospores form in abundance as the supplies of food and moisture get loosened. In artificial cultures, the *Fusarium* spores often grow singly from short lateral hyphae, not side by side on stromatic cushions of pseudoparenchymatous fungus tissue, as on the bark. In such cultures also, every intermediate stage as regards size, shape, and number of septa, may be found between the *Cephalosporium* and *Fusarium* forms (Fig. 98, 4).

On the roots of wilted plants of pigeon pea (as on dead roots of many other plants in India) it is not uncommon to find the bright red perithecia of the fungus *Neocosmospora vassinfecta*. On stems of wilted plants in Dehra Dun, the rather similar red perithecia of *Nectria Bobbophylli* frequently occur. It so happens that both these fungi possess lower stages corresponding so closely with the conidial forms of *Fusarium udum* as to be practically indistinguishable without the aid of cultural methods. It was only by isolating each of the three fungi in pure cultures and testing the parasitism of each separately that it was possible to establish *Fusarium udum* as the true cause of the disease. The case is a good one as illustrating the difficulty which is sometimes experienced in ascertaining the cause of crop diseases and the necessity of proving the parasitism of the suspected fungus by inoculation; the more so as *Neocosmospora vassinfecta* was previously believed to be the cause of a number of wilt diseases of crops in different parts of the world.

As already stated, infection probably always occurs through the roots. The fungus remains alive in the soil, even in the absence of the host plant, for a considerable period, certainly more than a year. The *Cephalosporium* and *Fusarium* forms of conidia are capable of germination when nearly a year old. The chlamydospores probably retain their vitality for a still longer period. The prevalence of the disease and the extent of damage caused by it, are largely due to soil infection from the preceding crop. The fungus passes from decaying roots into the soil and continues to grow and form spores under saprophytic conditions until the next crop is planted. It is not, however, capable of indefinite life in cultivated soils; otherwise it would be practically impossible to grow pigeon pea on a large scale in the infected areas. Under the normal rotation practised in Bihar, where pigeon pea is not usually again grown on the same land for three years, most of the fungus will have disappeared by the time the new crop is sown and only scattered cases of wilt will be found. Spread from these takes place chiefly through the soil, as the occurrence of gradually increasing patches of the disease clearly shows. The rate of this spread appears to depend on the space between the plants and the size of the latter; it is more rapid, the more crowded and the larger the plants are. This indicates that the fungus extends more readily along the roots than across intervening soil.

It has also been proved, however, that new cases of the disease may arise at a distance from the original patches, as a result of infection by

spores blown from the stems of wilted plants on to the soil. The fungus has been obtained from the surface of the stem well above the ground level, and the spores isolated from this position have been shown to be capable of causing the disease when added to the soil in which healthy plants are growing. This aerial-borne infection, however, usually occurs late in the season, as superficial spores are only produced in quantity freely exposed to currents of air when the wilted plants are of good size. Hence the damage caused in this way is not often considerable, being limited to single, scattered plants, which have not time to infect many of their neighbours before harvest. There is no evidence that the disease is conveyed in the seed, though, of course, it is quite possible that spores may adhere to the seed during harvesting operations, and reach the soil when the seed is sown. Experiments indicate that this is not a common method of spread.

Treatment of any disease which attacks plants through the roots is difficult. Obviously an attempt would have to be made to kill the parasite in the soil without injuring the roots; and this would have to be done early, as once the fungus enters the plant it is out of reach of external applications. Many experiments have been made with fungicides added to the soil, in the attempt to check various *Fusarium* wilts. Formalin injected into the soil was shown to be effective in checking carnation wilt in France, but the cost was about Rs. 300 per acre! In the United States, a large number of soil fungicides has been tried against cotton wilt, but without success. Tests with sulphate of iron and lime have been made in India, but were not effective in checking pigeon pea wilt. It does not appear very probable that any satisfactory method of disinfecting soil which contains parasitic *Fusariums* will be found.

Since direct treatment is not hopeful, indirect measures must be adopted. The chief of these is undoubtedly rotation, a sufficient interval being left between successive crops to allow the fungus to die out. In parts of Europe and the United States, it is found necessary to leave as much as six or eight years between successive crops of flax, on account of the allied flax wilt. The usually small holdings in India make it difficult to adopt really lengthy rotations, and where pigeon pea is of such major importance as a food crop as in Bihar, it is probable that the local practice could not be altered without serious inconvenience. On the whole, the disease is kept within reasonable limits by the rotation adopted, as it does not often destroy ten per cent. of the crop. Whenever it appears, the wilted plants should be systematically removed and

burnt, as they are a source of danger owing to the formation of spores on the surface of the stem.

The only thoroughly satisfactory way of dealing with wilt diseases is the selection or breeding of resistant varieties. In the United States, for instance, it has been found that Egyptian cottons are much less liable to cotton wilt than most of the local varieties ; while remarkable success has attended the attempt to produce resistant varieties by selection and hybridisation within the local races, not only in cotton, but also in water-melon, cowpea, and other plants subject to wilt and to certain other diseases. So also the cotton known as Buri in India is quite immune to cotton wilt in the Central Provinces. In spite of an extensive search, no variety of pigeon pea has been observed to resist the disease in this crop. Individual plants have, however, been found which appear to be more or less resistant, and an attempt is being made to isolate and propagate these. It is too early yet to predict what measure of success is likely to attend this method.

Wilt diseases similar to that of pigeon pea and probably caused by species of *Fusarium*, are known in India in cotton, gram, sesamum, sann-hemp, cowpea, *Hibiscus cannabinus*, and other crops. The general characters of these diseases resemble those described above, and their treatment will probably depend on much the same considerations. It will be necessary, however, to ascertain the cause and to work out the life-history of the parasite responsible in each case, before any accurate knowledge of the disease can be gained. From what is known of these fungi, it is probable that each is confined to a single, or at most a few closely allied hosts. The pigeon pea *Fusarium* has not been found on any other plant.

#### FIELD AND GARDEN PEAS (*Pisum arvense* L. and *P. sativum* L.).

**Downy mildew** (*Peronospora Viciæ* (Berk.) de B.).—This fungus occurs on the leaves of field and garden peas and is not uncommon in the Indo-Gangetic Plain. It forms a downy growth on the under surface of the leaves, in patches of varying size, sometimes covering most of the leaf surface, and of a greyish-violet colour. This growth is composed of conidiophores, arising from the mycelium of the fungus which is buried within the leaf tissues.

The mycelium consists of hyaline, unseptate, branching hyphæ, confined to the intercellular spaces except for branched, finger-shaped haustoria which penetrate into the cells.

The conidiophores arise directly from the internal hyphae and emerge in clusters from the stomata of the under surface of the leaf. They are long, unbranched for two-thirds or more of their height, then six to eight times bifurcated, the ultimate ramifications

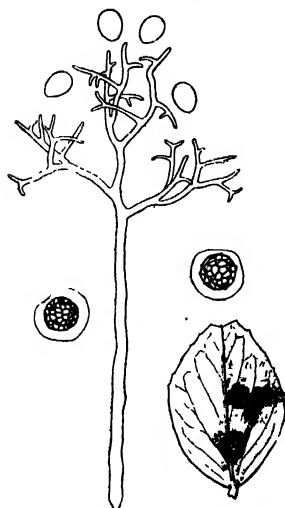


FIG. 99. Downy mildew of peas (*Pisum sativum*) leaf of field pea with mildew on under surface,  $\times \frac{1}{2}$ : conidiophore, conidia, and oospores,  $\times 200$ .

tions diverging from each other at a right, or more often obtuse angle and bearing conidia singly at the tips. The conidiophores measure 400 to 700 by 9 to 11 $\mu$  and all the branches are stiff and straight or only slightly curved. The conidia are nearly oval but narrowed a little below, pale violet in mass, and 22 to 27 by 15 to 19 $\mu$  in diameter. They germinate in moisture as soon as ripe, by a germ-tube which usually emerges from the side of the spore. As they are blown about freely by the wind, they serve to spread the disease rapidly from plant to plant, but they are short-lived and need the proximity of the host plant for further development.

In the old withered leaves oospores are found buried in the tissues. They are roundish, light brown bodies, with a thick episporium marked by a large, raised reticulation and measuring 28 to 32 $\mu$  in diameter. They lie singly within a larger thin-walled oogonium, which disappears after a time. The oospores germinate first in the season following that in which they were formed, and are very resistant to drying and other unfavourable conditions.

The disease is rarely very injurious to peas and treatment has not been resorted to in most countries in which

it occurs. To the crop *Lathyrus sylvestris* L., which is sometimes cultivated as a fodder in Central Europe, very great injury has been reported on several occasions. In this case, it has been found that much benefit results from cutting the crop at an early stage, before oospores have time to form. The plant grows again from the roots, and if the leaves have been carefully removed in the first instance, the new growth remains uninfected, since all the conidia have perished and oospores have not had time to ripen. Spraying with Bordeaux mixture has also been found effective in limiting the spread. Such active measures do not seem to be called for with Indian peas.

Besides the crops mentioned above, many allied plants, including lentils and various vetches, are also attacked by this parasite. In India it has been found on Khesari (*Lathyrus sativus* L.) in the Punjab and probably also occurs on lentils. The damage to these crops is slight.

**Powdery mildew** (*Erysiphe Polygoni* DC.).—This is a much more serious disease of peas than the downy mildew. It is usually late in developing, reaching its maximum intensity when the peas are in pod. It is also worst in dry years, unlike the downy mildew which is favoured by moist weather. Besides peas, the same fungus attacks beans (both broad and French), urid, lucerne, coriander, fenugreek, turnips, and many other plants belonging to several different families.

It is known in most parts of the world. Its prevalence in autumn in some districts of Britain has caused the growing of late varieties of peas to be abandoned; in the western United States also it is sometimes so destructive to late peas as to render their cultivation impossible. Early varieties are less injured, and at Pusa those that mature in January suffer little as compared with the February and March crops. Field peas are seldom badly attacked in India.

The fungus appears as white, floury patches on both sides of the leaves, as well as on the other green parts of the plant. These may cover large areas almost continuously. They consist of the mycelium and conidial stage of the fungus, the former being, as in most Erysiphaceæ, entirely superficial on the plant, sending only suckers (haustoria) into the epidermal cells.

The mycelium is generally fine and persistent, rarely thick. The haustoria develop as outgrowths from lobed swellings on the side of the hypha next the leaf; these are known as appressoria and seem to fix the mycelium to the leaf. The haustorium arises as an exceedingly narrow tube from the appressorium, which penetrates the cell wall and swells up into a rounded sac in the epidermal cell.

The conidiophores arise vertically from the leaf and each bears several spores in a chain; of these only the end one is mature, as they are formed in regular order from the tip backwards, and the ripe spores fall off quickly. The production of conidia is so copious



FIG. 100. Powdery mildew of peas (*Erysiphe Polygoni*): leaf with white mycelium dotted with perithecia, nat. size.

as to give the surface of the mycelium a characteristic powdery appearance. They are elliptical, barrel-shaped, or sometimes cylindrical cells, the last shape being confined to the forms occurring on certain host plants, not including the pea. They measure 25 to 35 by 13 to 16 $\mu$  and are colourless and unicellular as in the other oidial forms of the Erysiphaceæ.

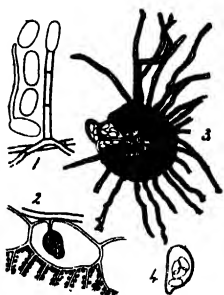


FIG. 101. *Erysiphe Polygoni*: 1, conidial stage from *Anthriscus sylvestris*,  $\times 180$ ; 2, haustorium from *Polygonum aviculare*,  $\times 225$ ; 3, perithecium from pea,  $\times 58$ ; 4, ascus with ascospores,  $\times 130$ . (1, 2, and 4 after Salmon.)

Later on, the perithecia appear as sharp black specks, scattered on the surface of the white mycelium. They are round, small (usually about 90 $\mu$  in diameter), composed of distinct polygonal cells, and with a variable number of "appendages," hyphæ which grow horizontally from the wall to a greater or less distance. These appendages may be few or many, dark coloured or colourless, and free or interwoven with one another or with the mycelial hyphæ. They are septate when coloured and are often angularly bent or flexed, but usually unbranched. Within the perithecium, a limited number of asci is formed, usually 2 to 8. These are usually ovate, nearly sessile, 46 to 72 by 30 to 45 $\mu$  in diameter, and contain 3 to 8 spores. The spores are elliptical, non-septate, colourless, and measure 19 to 25 by 9 to 14 $\mu$ . The perithecia with their contained ascospores persist in the soil, which they reach from the debris of fallen leaves, until the following season, when they disintegrate sufficiently to liberate the spores,

these then germinate and infect the new crop.

When the conidia germinate on a suitable host plant, the germ-tube early develops an appressorium at its tip, and from this a haustorium is sent into the underlying epidermal cell. With the food supply thus tapped, the superficial hyphæ continue their growth and develop new haustoria in constantly increasing numbers as they spread. When germinated in water on a glass slide, the appressorium forms as soon as the germ-tube comes in contact with the glass, but does not attempt to give out a haustorium. Hence it is suggested that mere contact with a hard substance induces appressoria to form, but that some further stimulus (? nutritive) is necessary to promote haustorium-formation.

Experiments have shown that this mildew has specialised races on its different hosts, as in the grass mildew or the cereal rusts (p. 61). The form on *Pisum sativum* (garden peas) has been found to attack *P. arvense* (field peas), but could not be got to infect the other hosts on which it was tried.

The treatment is similar to that of other powdery mildews (p. 146). A single application of sulphur dusted on the green parts has been found entirely to check spread. Spraying with Bordeaux mixture is also very effective.

**Rust** (*Uromyces Fabæ* (Pers.) de B.).—This rust is found in all its stages, spermagonia, æcidia, uredo, and teleuto, on peas, broad beans (*Vicia Faba*), and lentils in India (Fig. 14). In many other parts of the world it is common on these hosts but, in Europe at least, spermagonia and æcidia seem to be rare on the first two and unknown on the last. The rust also attacks several allied plants, mostly weeds.



The fungus is found during the early months of the year in the Indo-Gangetic Plain, being most common on peas in Pusa, though in 1911 it did some damage to lentils.

The aecidial stage appears first. Sporangia accompany the aecidial cups, which may develop on all green parts of the plant, but on the leaves are chiefly on the under surface. They appear in yellow spots, singly or in round or elongated clusters. The peridium is short, whitish, cup-shaped, and with an indented, reflexed margin. The spores are round-angular to elliptical, yellow, with fine warts, and 14 to 22 $\mu$  in diameter.

The uredo sori are on both leaf surfaces and on the stem and petioles, often arranged in little circles, small, powdery, and light brown. The spores are round to ovate, light brown, spiny, with 3 or 4 germinopores, and measure 20 to 30 by 18 to 26 $\mu$ .

The teleuto sori are similar but most often on the stem and of a darker brown, almost black colour. The spores are sub-globose, ovate, or elliptical, with the apex rounded or flattened and the wall darker and greatly thickened (up to 11 $\mu$  thick) at this point. They are smooth, brown, and 25 to 38 by 18 to 27 $\mu$  in diameter. The stalk is persistent on the tallon spore, pale yellowish-brown, thick, and up to 90 $\mu$  long. On germination, the usual 4-celled promycelium is developed from the tip and bears sporidia as in the cereal rusts; but unlike the latter these sporidia have been found to infect the same plant as that on which they were borne, and then give rise to the aecidial stage. As fresh infections may continue to occur from time to time the aecidial stage may go on up to quite a late stage in the life of the host plant.

The fungus has specialised races, most of which seem, however, able to attack the pea. The form on lentils is apparently more restricted.

The effect of the attack is sometimes to cause partial defoliation, but usually the injury seems to be slight. Treatment is not called for in India, so far as has been observed. Broad beans are said to be sufficiently damaged to justify spraying in other countries, but there is little evidence that spraying is effective. Sulphur dusting against the powdery mildew at Pusa, while checking the latter, had no marked effect on the rust, though it was prevalent at the same time. If the peas or beans can be picked early, much of the teleuto stage may be destroyed by cutting and burning the crop before many leaves fall. This will reduce the chance of infection the following year.

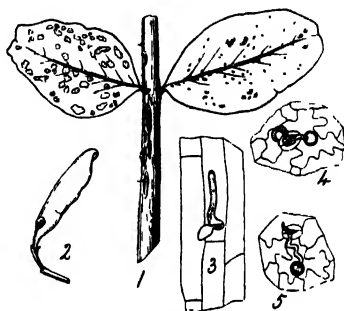


FIG. 102. Pea rust (*Uromyces Fabae*). 1, uredo and teleuto sori, latter chiefly on stem,  $\times \frac{1}{2}$ ; 2, pod with aecidial stage,  $\times \frac{1}{2}$ ; 3, germ-tube of sporidium entering through cells of bean stem,  $\times 200$ ; 4, germ-tube of aecidiospore entering through stoma,  $\times 95$ ; 5, same of uredospore,  $\times 95$ . (3-5 after de Bary).

In Europe, peas are attacked by a second Uromyces, *U. Pisi* (Pers.) Wint., which is heteroecious, the æoidial stage being found on Euphorbia. This species is not known in the East.

**BEANS:** FRENCH (*Phaseolus vulgaris* L.); URID (*Ph. Mungo* L. var. *Roxburghii* Prain); MUNG (*Ph. radiatus* L.); MOTH (*Ph. aconitifolius* Jacq.); RANGOON (*Ph. lunatus* L.).

**Powdery mildew** (*Erysiphe Polygoni* DC.).—Urid and mung are often attacked by this mildew in northern India and Madras, and it is known also on allied species of *Phaseolus* in other countries. As the disease has been described under peas, it need not be further referred to.

**Anthraxnose** (*Glomerella Lindemuthianum* (Saco. & Mag.) Shear: = *Colletotrichum Lindemuthianum* Br. & Cav.; *Glæosporium Lindemuthianum* Sacc. & Mag.).—The bean anthraxnose is known almost everywhere that French beans are cultivated, and in northern India and the Nilgiris sometimes does considerable damage in gardens.

It attacks all parts of the plant except the roots, from the primary



FIG. 103. Bean anthracnose (*Glomerella Lindemuthianum*): 1, affected pod,  $\times \frac{1}{2}$ ; 2, acervuli,  $\times 245$ ; and conidia,  $\times 480$ , of *Colletotrichum* stage; 3, germination of conidia showing appressoria. (J after Edgerton.)

shoot and cotyledons of the seedling to the leaves, stem, and especially pods of the mature plant. It is characterised by the appearance of black, rather sunken spots, with reddish or yellow, slightly raised margins. These begin as small brownish or purplish specks, which enlarge radially and darken in colour, until they may reach a diameter of more than a quarter of an inch. The infected tissues dry up and collapse, leading to the depression of the centre of the spot, which, at this stage, especially in moist weather, becomes pink and oily in appearance from the liberation of spores. The spots are usually round, but when two or more coalesce may become irregular; on the stem, they may cover several inches and cause the tissues to crack and rot away. On the pods, they are

most distinct and on opening the pod are often found to have penetrated to the inside and to have reached the seed. On the leaves, they are mostly

on the veins and petiole, on the lower side, and in young leaves may cause crinkling and distortion.

The mycelium of the parasite is localised in the tissues of the discoloured parts entering directly into the cells and rapidly killing them. The hyphae may penetrate right through the thickness of the pod wall and pass from its inner surface into the seeds inside, causing, as they ripen, the appearance of black, sunken spots on the surface of the seeds at the points of infection. As the mycelium becomes well nourished, it accumulates in and below the epidermis, and especially between the epidermis and the cuticle, raising up the latter into little pustules by the growth of a stromatic layer of fungal cells. From the surface of this layer, short, cylindrical stalks arise, densely crowded side by side. On the tips of these, the spores are formed singly, and by their growth the cuticle is ruptured and the surface of the spot becomes covered with pink masses of spores, held together by a mucilaginous secretion. The earlier spores are said to be formed directly on the hyphae near the surface, the development of the stroma occurring later. In some cases the circumference of each stroma is provided with stiff, erect hairs (setae), arising singly from the marginal cells, dark brown in colour, unbranched, and septate. These are never numerous and often entirely absent, and the conidial stage was variously referred to the genera *Colletotrichum* and *Gloeosporium*, according to the presence or absence of these hairs, before its aecigerous form was known. Recent work has shown that the species of *Gloeosporium* which are lower stages of the genus *Glomerella*, cannot be kept distinct from *Colletotrichum*, since the hairs which alone separate the two forms may be present or absent according to circumstances.

The spore stalks (basidia) are cylindrical, hyaline, unbranched, unseptate, and measure 45 to 55 $\mu$  in length. The conidia are hyaline singly, pale pink in mass, unicellular, varying from cylindrical to long oval in shape, with rounded ends, and sometimes very slightly curved. They measure 15 to 19 by 3.5 to 5.5 $\mu$  in diameter and germinate readily in water after liberation, sometimes becoming divided by a median septum and even swelling up into a dumb-bell shape when germination begins. Each conidium forms round it a sort of gelatinous layer which sticks the spores together; hence they are not readily disseminated until after rain, the water dissolving out the spores and washing them down to the soil. This probably explains the much greater frequency of the spots on the lower surface of the leaves and other parts liable to be splashed by wet soil, although the upper surface is just as easy to inoculate artificially as the lower. It also explains why spread from field to field or across paths and the like, is not common.

Germination usually cannot occur until the spores have been washed free from their mucilaginous bed; the germ-tube then grows out from near one end, less often from each end. When on the surface of the host plant, the germ-tube remains short and swells up at its end into a rather thick-walled, angular or roundish, brown enlargement, which is an appressorium of a less specialised type than that described in the *Erysiphaceae*, since it has other functions than merely to secure penetration of the epidermis. Unlike the latter, it is able to form a new hypha even when not in contact with the host and thus acts also as a chlamydo-spore. From the appressorium, a fine hypha arises on the side next the epidermis, which it pierces to ramify as a parasitic mycelium within the tissues. In cultures in water or on nutrient substances, these chlamydo-appressoria are freely formed not only on the germ-tube but also on the general mycelium; conidia of the normal type may also develop in these situations, as well as directly on the germinating spores, being borne singly on short stalks.

Perithecia have been obtained in culture. They are variable, globose to pear-shaped, with a very short or a long beak, usually sunken in the substratum, dark, and about 250 to 300 by 100 to 200 $\mu$  in diameter. The asci are cylindrical or enlarged above, thickened at the apex, up to about 70 $\mu$  long by 7 or 8 $\mu$  broad, and with 8 spores, usually

in two rows. The spores are cylindrical, curved, unicellular, hyaline or pale yellowish-pink (in mass), and 15 to 22 by 4.5 to 6 $\mu$  in diameter.

Successful inoculations have been carried out by sowing the conidia on healthy pods. New spots may appear at the infected points in 4 or 5 days, or even, according to some, in 24 hours. Continued moisture for about a day after inoculation is necessary to get the best results. There is still some doubt as to whether the present species is capable, like most anthracnose fungi, of living on several hosts. Some have claimed that it may be transferred to cucurbits, brinjal, and other plants, but most workers have failed to inoculate anything but beans. Pulses such as cowpea and val (*Dolichos*) are subject to an anthracnose in India which cannot be distinguished from that described above; and though cross-inoculations have not been tried, they have been for the present assumed to be identical (Fig. 108).

Carefully conducted experiments, especially in the United States, have proved beyond all question that seed taken from diseased pods into which the fungus has penetrated so as to infect the contained beans, gives rise to diseased seedlings. The mycelium of the fungus, in these cases, reaches the seed coats, the fleshy cotyledons, and even the embryo. If kept dry, it seems to cease growth until planting time; then, when germination occurs, it is carried up on the young shoot and resumes active growth, forming spores on the young stem and the cotyledons. These first-formed spores are washed to the soil and also pass to any of the new leaves that touch them. Spread is said to be chiefly by rain splashing, wind dissemination being of little account. All newly-developed parts of the plant may thus be infected in turn, if the atmospheric conditions are favourable to the parasite. Spread from field to field is slight and usually due to carelessness of the workers passing from one field to another. In some places, experience has taught the danger of working in the crop on wet days, the spores being carried about on clothes and implements.

Like so many other fungi that depend on moisture for successful infection, marked periodic variations in severity, depending on climatic conditions, are noticeable. Rain or high humidity when the crop is most actively in growth and also when the pods are forming, greatly increase the spread. The epidemic in the United States in 1906 followed a gradual increase, lasting through several years which were progressively wetter, and culminating in a very wet season in 1906. In each of the preceding years, a steadily increasing number of seeds were infected in their pods and, when sown, gave an increasingly large number of diseased seedlings. In the 1906 crop an average of 20 per cent. of diseased seed was found in a large number of examinations. In 1907, the season was exceptionally dry and, though most of the plants were spotted, the growth did not develop to any extent on the pods and the 1907 crop of seed was relatively free from the disease. Temperature is sometimes a controlling factor:

in the southern United States the disease is said to cease to develop when the mean temperature rises above 80°F. with the minimum above 70°F. Such temperatures are not, however, usual in the season when French beans are grown in India.

The perithecial form, which would serve to carry on the fungus through the period when the host plant is not growing, is probably very rare in nature. Most of the conidia seem to be short-lived but some have been germinated successfully from infected seed a year old. Experiments seem to show that dried spores lose their vitality in about a month, but if between the cotyledons in the seed they probably escape drying. The same may perhaps occur in the fallen débris of diseased plants in the soil ; but the evidence available indicates that the parasite is usually carried in the mycelial condition in the seed, as described above, and that early infection from spores is rare.

Hence the treatment is based on the elimination of all diseased seed. Attempts to do this by selecting for seed such beans only as show no sign of spotting, have not been very successful ; apart from the labour of hand-picking the seed, it has been found that many beans may be infected without external markings sufficient to enable them to be readily detected. On the other hand, diseased pods can always be easily known by the spots, and if all spotted pods be discarded and seed taken only from such as are clean, the resulting crop will tend to be free from primary infection. In the United States, seed taken from localities where the disease is absent, or picked from only such pods as are quite unspotted, has been found to give a crop free from anthracnose.

Several other methods of checking the disease have been advocated. Seed treatment by fungicides is useless, since the parasite is in the interior of infected beans and out of reach of the treatment. Spraying with Bordeaux mixture has given good results on a small scale, but when applied in the field, so many difficulties in reaching all parts of the plant have been encountered that it has proved ineffective in checking the attack sufficiently to be profitable. Burning the infected plants has been recommended in order to avoid any danger of the remains of diseased plants carrying on the fungus to the next crop. For the same reason, it is said to be unwise to grow beans on the same land in successive years. Resistant varieties of beans have been found and seem to be rather extensively grown in the United States but have not always proved satisfactory. It has been discovered that there are different strains of the parasite, and that a variety resistant to one strain may succumb to

another. In a given locality, the variety may preserve its resistance for some time, if the local strains of the parasite are not able to attack it; but other strains may be introduced, through seedsmen for instance, and the variety fails. On the whole, therefore, no method gives such good results as the use of clean seed.

The fungus has been found to attack the scarlet runner (*Phaseolus multiflorus*) and, to a less extent, *Ph. lunatus* (Rangoon bean). Whether the forms on other pulse crops are merely specialised races or distinct species is not known; but the existence of separate races on the French bean itself points to the probability of there being a more extended series of specialised forms on allied hosts subject to similar diseases.

**Rust** (*Uromyces appendiculatus* (Pers.) Lk.).—This fungus is parasitic on French beans, mung, and urid, as well as on cowpea, val, and one or two allied weeds, in India. Like the pea rust, it is widely distributed in parts of the world where these crops are grown, but it does little damage on the whole, much less than the anthracnose just described.

It resembles *Uromyces Fabæ* in having all four stages on the same host. It differs chiefly in the characters of the teleutospori, which in *U. Fabæ* is much thickened at the apex, while in *U. appendiculatus* the wall is of almost equal diameter all round, the apex being only marked by a broad, pale papilla. The teleutospore stalk also is shorter in the present species.

The point of attack is usually on the leaf, rarely on the stem and pod. The mycelium is localised near the seat of infection and, like other rusts, occupies the spaces between the cells, only sending haustoria into the cell cavities. The acedial stage is not frequent and resembles that of *U. Fabæ*, except that the spores are bigger, 18 to 36 by 16 to 24 $\mu$ , and often rather oblong. It is followed by the uredo sori, which are usually found on the under surface of the leaves as small, powdery, brown pustules. They are developed under the epidermis, which is ruptured to liberate the spores. The latter are round to ovate, light brown, spiny, with 2 germ-pores, and measure 18 to 28 by 18 to 22 $\mu$ .

Teleuto sori appear later on, chiefly on the under surface of the leaves, but also sometimes on the upper surface. They form small, compact, black warts

which, on the upper surface, are surrounded by a yellow band. The teleutospores are sub-globose, ovate, or elliptical, rounded at the apex which has a large hemispherical

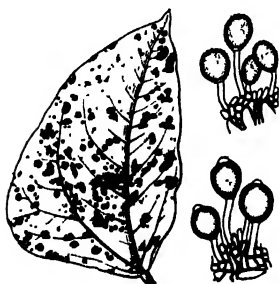


FIG. 104. Bean rust (*Uromyces appendiculatus*): leaf of cowpea with sori,  $\times \frac{1}{2}$ ; uredo and teleutospores from cowpea,  $\times 100$ .

hyaline papilla, smooth except sometimes for a few small warts towards the top, chestnut-brown, and 24 to 35 by 18 to 25 $\mu$  in diameter. The stalk is about equal to the spore in length.

The teleutospores preserve their vitality in the soil débris until the following season, when they germinate and bear sporidia which infect the new crop.

The remarks on treatment under pea rust apply equally here.

The search for resistant varieties is promising, since one has already been discovered in the cowpea. This is the variety known as "Iron," now extensively cultivated in the southern United States because it is the only kind which escapes damage from wilt and root-knot.

**Leaf spot** (*Cercospora cruenta* Sacc.).—This fungus was first described from the United States on a species of *Phaseolus* or *Dolichos*, and has since been recorded in America on French beans, Rangoon beans, and cowpea. In Italy it has been found on *Dolichos sesquipedalis* and in India on French beans, mung, and moth. A second species was subsequently described on cowpea in the United States as *Cercospora Dolichi* E. and E. and the common leaf spot of *Dolichos* in India has been identified as this. There is, however, no constant difference between the two and they are probably identical. Two other forms, *C. canescens* E. and M. on a cultivated *Phaseolus* and on Rangoon beans in the United States and on mung in China, and *C. Vigna* Rac. on cowpea in Java, are also probably the same as *C. cruenta*, which would thus appear to be a very widespread parasite.

The leaf spot is found on both rains and cold-weather pulses, and

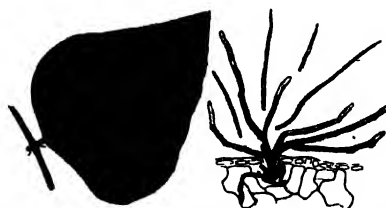


FIG. 105. Bean leaf spot (*Cercospora cruenta*): leaf of French bean with spots,  $\times \frac{1}{2}$ ; conidiophores and conidia,  $\times 90$ .

attacks young and old plants. The leaves are marked with spots which are at first small and brown or red, then grey or brown in the centre with (on most hosts) a prominent reddish-purple border. The spots are usually angular, from being limited in their spread by the veins of the

leaf, and sometimes the centre breaks away, leaving a hole. Severe attacks cause the leaves to be shed and a good deal of injury may be caused by weakening the plant and checking the formation of pods. The stems are also occasionally attacked.

The conidiophores emerge in clusters through the stomata. They are brown, septate, with knee-bends marking the insertion of fallen spores, as is usual in the genus, and about 50 to 100 $\mu$  long by 4 to 6 $\mu$  broad. The conidia are borne terminally, but the stalk continues to grow at one side so that the spore is pushed over and falls off. They are variable in size, from 50 to 140 by 4 to 5 $\mu$ , elongated, tapering above, with 2 to 8 septa, straight or slightly curved, and almost colourless.

Different races of the host plants seem to vary in their susceptibility to the disease and probably the best way to reduce the damage is to develop disease-resistant varieties. The hardy "Iron" cowpea is relatively immune in the United States. Spraying and other direct treatments are not likely to repay cost.

### COWPEA (*Vigna Catjang* Endl.).

**Rust** (*Uromyces appendiculatus* (Pers.) Lk.).—(See p. 260.)

**Powdery mildew** (*Erysiphe Polygoni* DC).—(See p. 253.)

**Anthracnose** (? *Glomerella Lindemuthianum* (Sacc. and Mag.) Shear).—The anthracnose of this crop agrees in general with that of the bean, but experiments are necessary before they can be pronounced identical. It is possible that the parasite may prove to be the more common *Glomerella cingulata* (pp. 448, 512), there being no essential microscopic differences between the two.

**Die back** (*Vermicularia Capsici* Syd.).—A fungus agreeing with that which causes the die back of chilli (p. 352) has been observed at Pusa associated with dying back of the stems and shrivelling of the pods. Successful inoculations were obtained with the parasite from chilli on cowpea, val, tomato, and brinjal, showing that it has a considerable range of hosts. It is fully described under chilli.

**Root rot** (*Hypochynus Solani* Pril. and Del. = *Rhizoctonia Solani* Kuehn; and *Rhizoctonia* sp.).—The cowpea is attacked by at least two species of *Rhizoctonia* in India. Many other plants are subject to similar diseases but, as there is much confusion in the literature and nomenclature of *Rhizoctonia*, it is not yet safe to refer the majority of the Indian species to previously named forms. It is probable that there is little difference in the characters of the diseases caused by various species of *Rhizoctonia* and only the two fully studied on the cowpea need be referred to here.



The commonest form in India is one which produces small, black sclerotia, which is known as a parasite of jute (Fig. 7), cotton (Fig. 155), groundnut, cowpea, mung, Rangoon bean, val, kukthi, tomato, potato, brinjal, cucurbits such as *Citrullus* and *Cucurbitaria*, tobacco, lucerne, sesamum, and several other plants.

On the cowpea, affected plants are found with the upper portion brown and withered, while the stem at the ground level is blackened. In the early stages of infection, a red-brown discoloration appears on the stem and slowly spreads, causing it ultimately to become flaccid and collapse. At a later period, individual branches of the creeping stems may take the disease at any part, either by direct infection from the soil or by contact with a previously diseased part. In artificial inoculations, the growing point seems peculiarly liable to infection.

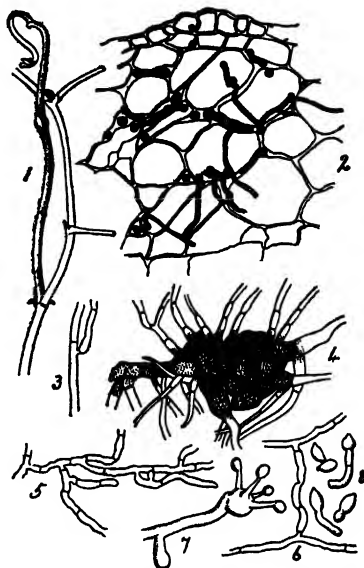


FIG. 106. Root rot (*Hypochnus Solani* and *Rhizoctonia*): 1, small sclerotial *Rhizoctonia* on dead branch of cowpea; 2, mycelium of same in outer cortex,  $\times 190$ ; 3, hyphae of same from cotton,  $\times 125$ ; 4, early stage of sclerotium-formation of same from cotton,  $\times 190$ ; 5, branching hyphae of *Hypochnus Solani*,  $\times 125$ ; 6, anastomosis of two hyphae of same,  $\times 125$ ; 7, basidium of same from cowpea showing four sterigmata,  $\times 330$ ; 8, basidiospores of same and their germination,  $\times 250$ .

Soon after the blackening of the tissues is well established, great numbers of tiny, black dots can be seen at the surface and in the outer layers of the tissues (Fig. 106, 1). These are the sclerotia characteristic of the fungus *Rhizoctonia*.

Microscopical examination shows that the tissues of infected parts contain, especially in the outer zones, numerous hyphae ramifying in all directions. In the early stages these hyphae are found in the cortex, chiefly between the cells (Fig. 106, 2). Should the infection be strong, intracellular hyphae are speedily formed and the inner tissues are penetrated.

The hyphae are colourless when young and abundantly branched, the branches usually arising almost at a right angle from the parent hypha, then often bending rapidly to lie nearly parallel to it. At the base, the branch is usually constricted and a cross wall ordinarily develops about  $15\mu$  from this point (Fig. 106, 3). The diameter of the main hyphae is about 8 or  $9\mu$  and they are divided by transverse septa at distances of from 50 to  $150\mu$ . The older hyphae, especially in artificial cultures, tend to turn brown; at a late stage, large, brown hyphae composed of short, barrel-shaped cells develop. These also are usually found at the beginning of sclerotium-formation.

The sclerotia usually appear as the tissues dry up. They are formed from a single hypha in which a large number of transverse divisions take place, so that a row of short, barrel-shaped cells is produced, very rich in oil globules. Some of these give rise to lateral outgrowths consisting of one or two cells, which grow parallel with, and adhere to, the side of the parent hypha. By the constant repetition of this process a mass of parenchymatous cells is formed (Fig. 106, 4), which eventually becomes the mature sclerotium; there is some slight differentiation into cortex and medulla, the central cells being large and thin-walled, while the peripheral cells have a smaller lumen and thicker walls (Fig. 7). Mature sclerotia are smooth, black, and measure about 100 to  $150\mu$  in diameter. They remain sterile so far as is known, no spore form having been found to develop on or in them. Their vitality seems to be variable; it is said that infected fields retain the fungus in the soil for as much as three years, and from their structure it is to be presumed that the sclerotia keep their vitality longer than the mycelium. Cultures have been found alive after six months.

In this species of *Rhizoctonia* it is not known with certainty to what genus, or even to which of the great groups of fungi, the perfect stage, if one exists, belongs. No spore form has been discovered, but there is reason to believe from the similarity to the species next to be described and also from the occasional occurrence of "clamp-connections" in the hyphae, that it is the sterile condition of a Basidiomycetous fungus, allied to *Corticium* or *Hypochnus*.

*Hypochnus (Corticium) Solani* Pril. and Del. is the name applied to a second parasite of cowpea, which, in its most commonly found sterile condition, is a *Rhizoctonia* with many points of similarity to the last.

It has been found in India on cowpea, groundnut, potato, tomato, and *Trichosanthes*, causing a disease characterised by the withering of the whole or part of the attacked plants. Seedlings of cowpea can be readily infected on the stem and at the growing point, but in the field the attack usually begins at the ground level. The infected parts turn a reddish-brown colour and the tissue becomes soft, the whole plant finally collapsing. On older parts, the attack may occur in any part, even on mature leaves, causing a withering in which the dried-up parts take a deep brown colour.

At a later stage, a second form of growth is found on some plants. In these, the leaves and green stems are coated with a white layer. This is the perfect form of the fungus.

The hyphae of the parasite are found abundantly in the diseased tissues. They closely resemble those of the last species, being from 6 to  $10\mu$  in diameter and divided by transverse septa into cells 100 to  $200\mu$  long (Fig. 106, 5). The branching is similar to

that of the other species and union of lateral branches from two hyphae sometimes occurs (Fig. 106, 6). Fine hyphae, such as are found in cultures of the other *Rhizoctonia*, are not present in this species. In old cultures, the hyphae turn a reddish-brown colour. Mature hyphae often are formed of short, broad, somewhat triangular cells; this is usually an early stage in the formation of a sclerotium.

The sclerotia are much larger than in the last species, varying from 1 to 5 mm. in diameter. They are formed on the surface of diseased parts, stems, leaves, and pods. The young ones are visible to the naked eye as fluffy aggregations of hyphae, at first quite white; later, the colour changes to brown and they become hard and compact. The mature sclerotium consists of interwoven hyphae, originating by copious branching from a number of different hyphae, not from a single one only as is usual in the other species. The ultimate condition is the formation of a large uniform mass of parenchymatous cells, without any differentiation into cortex and medulla such as is found in the other form. The sclerotia remain sterile, no spores being formed on or in the hyphae of which they are composed.

In the second form of growth, the white layer on the surface of the leaves and green stems is found under the microscope to consist of closely interwoven hyphae of the usual *Rhizoctonia* type. Where this layer is best developed, certain peculiar lateral branches can be noticed. These are short, club-shaped structures, with a transverse wall some distance from the point of origin. On the swollen end are found two to four finger-like processes (sterigmata), each of which carries a single spore (Fig. 106, 7). The club-shaped branches are typical basidia, proving the fungus to belong to the great group of the *Basidiomycetes*. The spores are hyaline, oval, unicellular, and about 10 to 12 by 6 to 8  $\mu$  in diameter (Fig. 106, 8). Under the basidial layer, the tissues are freely penetrated by hyphae of the type described above.

That this spore-bearing form belongs to the *Rhizoctonia* with large sclerotia has been proved by cultures, each type being capable of giving rise to the other. Apparently the basidial form is strictly parasitic, as it has not been found possible to produce it in cultures on artificial media, or anywhere but on living plants. In the field, it is found most vigorously developed on those parts which are well shaded and also in damp situations. After the spores have been shed, it wears off.

Both these fungi are soil dwellers and commence their attacks usually at or below the ground level. Both may occur together, and on this account they are often confused with one another. This is of little practical importance, as the same considerations in regard to lessening the damage caused by them apply to both.

Treatment should aim at destroying the fungus in the soil, where it persists no doubt mainly by means of its resistant sclerotia. It is probable that any direct attempt to rid the soil of the parasite is impracticable under ordinary conditions of extensive cultivation, and should be restricted to experimental farms and the like, where small plots of improved varieties sometimes have to be guarded from loss. Naphthalene and sanitary carbolic powder seem to be effective in preventing attack but in some cases (jute) the former seems to injure germination, and preliminary tests should be made in any case where soil disinfection with this substance is necessary.

Probably the best means of combating the disease is by a careful rotation of crops. The same considerations must guide us in this case as in the much more destructive *Fusarium* wilts. *Rhizoctonia* is not often truly epidemic, so far as experience in India goes, the cases being usually sporadic; but there is little doubt that want of proper cultivation and rotation could easily induce severe attacks. On the other hand, the fungus, like most soil fungi, fares badly in the presence of a good cultivator.

Experiments carried out at Pusa with strains of the small black form isolated from cowpea, cotton, groundnut, and jute show that with the first three, each can attack the other two but the attack is usually less virulent than with their own strains; the strain on jute cannot ordinarily infect cowpea, groundnut, or cotton at all.

### SOY BEAN (*Glycine hispida* Maxim.).

**Downy mildew (*Peronospora Trifoliorum* de B.).**—This fungus is well known in other countries as a parasite of clover and lucerne, but in India proper has only been found on some wild species of *Melilotus* and *Medicago* in the Indo-Gangetic Plain. In Kashmir, however, it attacks the soy bean, on which it has also been reported from Formosa.

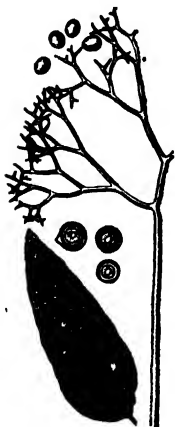


FIG. 107. Downy mildew of soy bean (*Peronospora Trifoliorum*): leaf with spots,  $\times 4$ ; part of conidiophore, conidia, and oospores,  $\times 130$ .

The characters of the disease are similar to those of the downy mildew of peas (p. 251), the leaves bearing a grey violet, downy growth on their under surface, the upper surface being correspondingly bleached. Affected leaves wither and drop prematurely.

The fungus agrees in its general characters with *Peronospora Viciae* on peas. Its specific distinguishing characters are as follows:—Conidiophores several together from the stomata, 300 to 450 $\mu$  long, 9 to 11 $\mu$  broad toward the base, branching 6 to 8 times near the top, the primary branches straight or but little curved, the intermediate branches more often curved and diverging at a right angle, the end branches lightly curved or nearly straight, diverging at a right or obtuse angle, pointed and bearing a conidium at each tip. The conidia are broadly elliptical, 18 to 24 by 15 to 18 $\mu$ , and pale violet. The oospores are immersed in the leaf, round, 24 to 31 $\mu$  in diameter, and

with a thick, smooth, light brown wall.

A variety of the fungus has been described on the present host from Russian Manchuria (*P. Trifoliorum* var. *manshurica* Naum.). It is said to have conidiophores twice

as long as given above (240 to 900 by 7 to 9 $\mu$ ), with nearly round conidia (21 by 1 $\frac{1}{2}\mu$ , 24 by 21 $\mu$ , 27 by 24 $\mu$ ), and large oospores (36 to 48 $\mu$  in diameter). The Kashmir fungus agrees more nearly with the normal type.

Treatment has not been tried on soy beans. The destruction of the oospores by burning the débris of diseased plants should be useful.

**VAL and KULTHI** (*Dolichos Lablab* L. and *D. biflorus* L.).

**Rust** (*Uromyces appendiculatus* (Pers.) Lk.).—(See p. 260.)

**Anthraenose** (? *Glomerella Lindemuthianum* (Sacc. and Mag.) Shear.) (Fig. 108, 1—2).—(See under Bean (p. 256) and Cowpea (p. 262).)

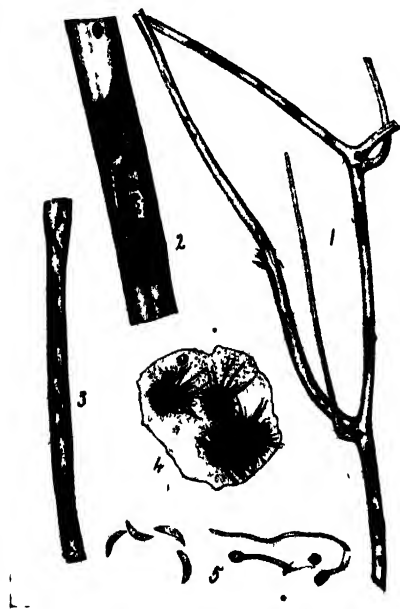


FIG. 108. *Dolichos anthracnose* (? *Glomerella Lindemuthianum*) and die back (*Vermicularia Capsici*): 1, anthracnose of stem,  $\times \frac{1}{2}$ ; 2, the same,  $\times 2$ ; 3, die back of stem,  $\times \frac{1}{2}$ ; 4, sporodochia of *Vermicularia Capsici*; 5, conidia of same and germination,  $\times 240$ .

**Die back** (*Vermicularia Capsici* Syd.) (Fig. 108, 3—5).—(See p. 352.)

**Root rot** (*Rhizoctonia* sp.).—(See p. 262.)

**KHESARI** (*Lathyrus sativus* L.).

**Downy mildew** (*Peronospora Viciae* (Berk.) de B.).—(See p. 251.)

**Rust** (*Uromyces* sp.).—The rust of this crop appears to have been only once collected, at Dumraon, Bihar, many years ago. The specimens cannot be traced, so that beyond the statement that the fungus resembles the rust of gram (p. 271), with which it agrees fairly closely in microscopic characters, there is no further information available. It is evidently rare and of little importance.

**LENTILS** (*Lens esculenta* Moench).

**Rust** (*Uromyces Fabae* (Pers.) de B.).—(See p. 254.)

**VELVET BEANS** (*Mucuna* and *Stizolobium* spp.).

**Rust** (*Uromyces Mucunae* Rabenh.).—Velvet beans (Florida beans) have been introduced in several experiment stations in India, and at Pusa give a very heavy growth which, however, has been checked to some extent by the copious development of this rust. It was first described from Calcutta in 1878, and has since been collected on wild species of *Mucuna* in several parts of India and Burma. More recently, it has been

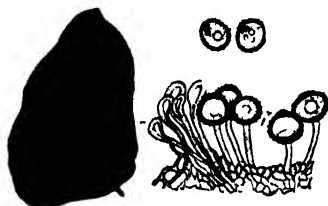


FIG. 109. Velvet bean rust (*Uromyces Mucunae*): leaf of *Stizolobium Derringtonianum* with sori,  $\times \frac{1}{2}$ ; part of a teliosorus with two uredospores above,  $\times 250$ .

found in the Philippines, Ceylon, and Formosa.

Only the uredo-telios stages are known. The uredospores occur mixed with the teliospores and are usually comparatively scanty. They are roundish, with dense pointed warts, almost colourless, and 18 to 22 $\mu$  in diameter. Curved, hyaline paraphyses, swollen at the top, accompany the uredospores. The teliospores are on the under surface of the leaves, scattered in large numbers as deep brown or almost black, powdery, raised cushions. The spores are round or globoid, covered everywhere with pointed warts, chestnut-brown in colour, 18 to 22 $\mu$  in vertical diameter, 18 to 25 $\mu$  across, with a thick wall and a hyaline, longish stalk.

No method of checking this disease is known.

**GRAM** (*Cicer arietinum* L.).

**Blight** (*Mycophthora pinodes* (B. and Blox.) Nüssl. = *Ascochyta Pisi* Lib.).—This fungus was reported as causing much damage to gram in the North-West Frontier Province in 1911. In other countries it is chiefly

known as a troublesome parasite of peas and can also attack vetches. The species on French beans, sometimes confused with it, seems to be distinct. Damage to gram has been recorded in Europe and Canada.

All the green parts of the plant are attacked, the symptoms recalling those of the anthracnose of beans (p. 256). Dark spots appear on the stem and leaves first, then on the pods. On the stem, they are oval or elongated; on leaves and pods, round and up to half an inch across. When well developed, the margin is brown and the centre yellowish, withered, and studded with the tiny, dark fructifications of the fungus, often concentrically arranged. In bad cases, they may surround the stem, causing the parts above to wither. As the attack frequently begins at the ground level, this condition may result in the death of the whole plant. The young shoots near the tip are also very liable to infection and may be killed back.



FIG. 110. Gram blight (*Mycosphaerella pinodes*): affected pods and stem; below, section of a pycnidium of *Ascochyta* stage.

When the plant survives long enough to produce pods, the spots develop on these with great vigour. In this case, as in bean anthracnose, the fungus penetrates into the cavity of the pod and reaches the contained seeds. Every stage may be found between that in which the seeds are altogether prevented from forming and that in which they mature successfully and are merely marked by a minute, brown patch on the surface. In some cases, even the brown patch may be absent when the pod is opened and will only show if the seed be exposed to damp for some days. In other cases, the seed coat is dried up and splits, exposing the folded cotyledons (seed leaves) below. Prior to germination, the fungus is often confined to the seed coat; but with the moisture during germination it renews activity and passes into the seed leaves, the young stem, and even the first roots.

The hyaline hyphae of the parasite are found in and between the cells of the tissue and numerous pycnidia are produced at the surface in the central, dead portion of the spots. The pycnidia are round or flattened, immersed in the tissues, and 100 to 200 $\mu$  in diameter, the individual cells of the wall being 5 to 7 $\mu$  across. Their colour is reddish-brown, and there is a round mouth opening to the surface. The spores are hyaline (pale pink in mass), oblong, obtuse at the ends, with a median septum, and measure 9 to 20 $\mu$  (usually 12 to 16) by 3 to 6 $\mu$ . Occasionally there are two septa. When young, there is no narrowing at the septum, but as the spore begins to germinate, it swells up and is deeply constricted in the middle. As in the bean anthracnose, there is a mucilaginous substance round the spores which assists in their expulsion from the pycnidium by swelling up when wetted, and fixes them in a crust around the mouth as they dry. On germination, one or two germ-tubes are put out (later, others often appear) and the young hyphae frequently anastomose. New pycnidia may appear in culture in about a week.

The ascigerous stage has been found to be not uncommon on the old stems and pods of peas attacked by this blight in the United States. It has not been seen in India. The perithecia are round, brown, 100 to 300 $\mu$  in diameter, and sunk in the tissues except for the upper part, which forms a more or less elongated neck with an opening at the tip. The asci are clustered at the base of the perithecium, measure 60 to 80 by 12 to 16 $\mu$ , and are thickened at the tip, which is provided with a pore through which the spores are liberated. The spores are 8 in each ascus, hyaline, elliptical-ovate, 2-celled, and constricted at the septum; the upper cell is slightly the larger and the ends rather obtuse. They are 12 to 15 $\mu$  long by 6 to 8 $\mu$  broad. They germinate in much the same manner as the pycnosporae, and the mycelium from them may bear ripe pycnidia in 6 days.

Inoculations have been carried out on peas and show that both the ascospores and the conidia cause typical spots to appear in about 5 days, and pycnidia very soon after. Species of *Vicia* are attacked with some difficulty and French beans not at all or only so as to cause insignificant marks.

The disease originates in the new crop in exactly the same way as bean anthracnose (p. 256). The germination of badly infected pea seed has been found to be from 6 to 20 per cent. only, but those slightly attacked germinate better. The fungus passes up on the young shoot, which it permeates, extending even into the first leaves. The seedling may be killed or only stunted, but in either case may infect plants in the neighbourhood, if the weather is favourable to spore production. In addition to this, there seems to be good evidence that soil infection also occurs, probably from the ascigerous stage formed on dead parts of the previous crop and reaching the soil with the plant debris. It has been found that peas planted on the same soil for two or more years in succession often suffer heavily, while two years' freedom from a susceptible crop is enough to clean the soil.

Hence the treatment should be exactly the same as that described above for bean anthracnose, with perhaps rather more attention paid to rotation.

Gram is subject to another disease in northern India which might be confused with some forms of the present blight, but in which no spots



appear, the plants withering off uniformly. It is a wilt disease, apparently allied to that of pigeon pea.

**Rust** (*Uromyces Ciceris-arietini* (Grogg.) Jacz. and Boy.).—The gram rust is common at times in India (Bengal, Bihar, Bombay, and Madras), but is very irregular in its appearance at Pusa, rare some years, abundant others. It is known also in Europe and East Africa.

The rust appears first chiefly on the leaves as small, round or oval, cinnamon-brown, powdery pustules, which tend to coalesce. These occur on both surfaces of the leaves, most frequently beneath.

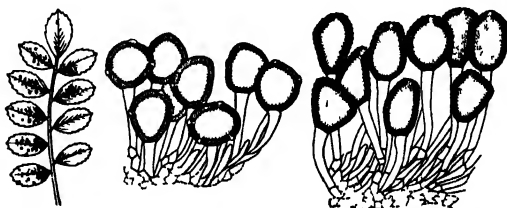


FIG. 111. Gram rust (*Uromyces Ciceris arietini*): leaf, nat. size; part of uredo and teleuto sori,  $\times 375$ .

Most of the pustules are uredo sori, which contain nearly round, brownish yellow to cinnamon uredospores, measuring 20 to  $28\mu$  in diameter, with scattered, minute spines or warts, and 4 to 8 (generally 4) germ-pores. They remain capable of germination for at least a month.

The teleuto sori resemble the uredo sori but are darker brown. The spores are variable in shape, round, ovate, or angular, with a roundish, unthickened apex, and brown, warty, or roughened wall. They measure 18 to 30 by 18 to  $24\mu$  and have a short, hyaline stalk, from which the spore readily becomes detached. They are not easy to distinguish from the uredospores, except in being somewhat longer and with only a single apical germ-pore.

The life-history of this rust is unknown. No æcidial stage has been found. Treatment is not practicable on present knowledge, but resistant varieties should be sought for.

#### GUAR OR CLUSTER-BEAN (*Cyamopsis psoraloides* DC.).

**Mildew** (*Oidiopsis taurica* (Lév.) Salm. = *Erysiphe taurica* Lév.).—The mildew of this crop occurs in Bombay and Madras, and the same fungus is known to attack several other plants in various parts of India. The number of its hosts is considerable but most are weeds, chilli, garlic, brinjal, fennel, and fenugreek, being the cultivated plants so far

found affected by it in India. It is widely distributed in south and central Asia, north Africa, and south and central Europe, but is rare in America.

Unlike all the other members of the Erysiphaceae, this species at first is entirely

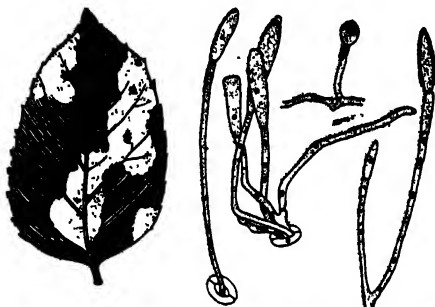


FIG. 112. Powdery mildew of guar (*Oldiopsis taurica*): leaf with white mycelial patches dotted with perithecia,  $\times \frac{1}{2}$ ; conidiophores and conidia (after Foer), the dwarf form in centre above is from the superficial mycelium.

endophytic. The septate, irregularly swollen hyphae are found in the earlier stages ramifying between the mesophyll cells of the leaf. They are abundant just below the cells of the epidermis and especially in the sub-stomatal spaces, but extend also between the cells of the spongy and palisade parenchyma, sending small, round haustoria into the cells, except those of the epidermis.

From this internal mycelium, conidiophores are early sent up through the stomata to the exterior,

singly or several in a bunch. The conidiophore arises at or near the end of an internal hypha and is thin-walled, septate, variable in length but often long (up to  $700\mu$ ), and usually branched. The branching may be more or less regular and each branch may end in a conidium; or branches may arise laterally from the stalk and spread indefinitely over the surface of the leaf to form an external mycelium like that of the other mildews.

Conidia are formed at the ends of the conidiophore and its branches, a single conidium at each tip. A second one may develop after the first has fallen. They are often very large, single-celled, cylindrical or irregularly shaped, hyaline, and measure 35 to 82 by 12 to  $28\mu$  in diameter. These extremes of size are not all found on the same host plant and the shape also varies to some extent according to the host: sometimes cylindrical spores with rounded ends predominate, sometimes ovoid or oblong with both ends rounded or with the apex narrowed to a point, while the middle part of the spore may be swollen or constricted. The wall may be quite smooth or (especially when old) rough with protuberances due to the breaking up of the episore.

Germination occurs with the production of a long germ-tube, which creeps over the surface of the leaf and forms one or more lobed (or even shortly branched) appressoria. Similar appressoria are found on the superficial mycelium but haustoria are not developed from them and the fungus appears to be nourished entirely from the internal mycelium. Infection takes place by the entry of the germ-tube into the leaf, probably through a stoma.

At a later stage, the mycelium accumulates on the leaf surface and resembles in appearance that of the other members of the Erysiphaceae; large numbers of hyphae emerge from the stomata to form an external, sometimes quite dense growth. Very rarely conidiophores are produced by this external mycelium. They are much shorter than those from the inside of the leaf, measuring only 50 to  $90\mu$  and consisting of three or four cells, of which the apical alone becomes a small, roundish, or oval conidium. From the

external hyphæ, new branches are again, at a later period, sent through the stomata to feed in the inside of the leaf.

On the drying leaves, the final or perithecial stage of the fungus appears, arising on the superficial mycelium, when this has drawn large stores of nourishment from the leaf cells. At this period, the mycelium is usually pretty dense and the perithecia appear as black specks, immersed more or less deeply in it. They are large (up to about  $250\mu$  in diameter), at first round, then cup-shaped from the falling in of the upper part, and furnished with numerous appendages or hyphæ springing from the perithecial wall. These are colourless or brown, more or less branched especially near the tip, interwoven, and rather short or sometimes even absent. The perithecium contains usually about 20 asci, which are large, cylindrical to ovate, usually long-stalked sacs, measuring up to  $110$  by  $25$  to  $40\mu$ , and containing each two spores. The ascospores are variable in size, from  $25$  to  $40$  by  $14$  to  $22\mu$ , but most often about  $32$  by  $18\mu$ , hyaline, unicellular, long oval, and sometimes slightly curved.

Attacked plants are defoliated and weakened by the premature drying up and death of infected leaves.

No doubt the parasite is transmitted from season to season by the perithecia, which in this family do not break up to liberate their enclosed spores until the wall has gradually rotted away. The fungus is a lover of warm or dry climates and its endophytic habit of life is probably an adaptation to the hot, dry climate of the localities where it is most abundant; such conditions must be unfavourable to the life of the more purely superficial species.

Treatment does not appear to have ever been tried. There is reason to fear that the internal position of the fungus in the leaf during part of its life would militate against the success of the remedies usually employed to check the Erysiphaceæ, such as sulphur dusting and spraying with potassium sulphide. Spraying with Bordeaux mixture is likely to be more satisfactory, as it would tend to prevent the spread of the parasite to sound leaves and its peculiar adhesive properties should preserve the leaves from infection long enough to avoid undue weakening of the plant. The sulphur remedies (except lime-sulphur) are more evanescent and unless they can kill the parasite outright, which is unlikely in this case, are of little value.

**Leaf spot** (? *Alternaria Brassicæ* (Berk.) Sacc.).—Guar is sometimes attacked at Pusa and in Madras by a leaf disease which appears to be identical with that of the Brassicas (p. 300). No morphological difference can be detected between the fungi which cause the two diseases, and the characters of the spots are quite similar in both cases. There is as yet, however, no evidence that the damage to guar is as considerable as it often is to the Brassicas, and a full account is postponed until we come to deal with the latter crops.

## CHAPTER VIII.

### VEGETABLES, ROOT-CROPS, AND OIL-SEEDS.

#### POTATO (*Solanum tuberosum* L.).

**Ring disease or bacterial wilt** (*Bacillus solanacearum* Smith).—This is the most prevalent potato disease in several parts of India, especially Bombay and Mysore. It has also been recorded in the United Provinces, the Nilgiris, and probably Bengal. For some years after 1891 it attracted much attention in Bombay, but an accurate account of it was first published in Mysore in 1909. More recently, the same parasite was found to cause the tobacco wilt in Bengal, and as the technical details regarding the characters and life-history of the organism were worked out in connection with the latter crop, a full account of them will be reserved for the section dealing with tobacco diseases. The parasite is of almost world-wide distribution, being found either on potatoes or tobacco in the United States (where it was first described scientifically in 1896), the Dutch Indies, Japan, and probably also Europe, South Africa, Ceylon, Malaya, Australia, and New Zealand, though the accounts are often too vague to permit of certain identification.

The disease is easily recognised by the sudden wilting of the plant, the leaves hanging flaccid as if the stalk had been cut through. Usually a few lower leaves are first affected, but in a few days the whole plant dries up. The tubers show the symptom from which the name is derived. If cut through, a brown ring will be noticed, beginning from the point of attachment on the underground stalk and extending round more or less completely to the opposite side. The course follows the ring of vascular tissue which encircles the tuber a little way below the rind. On squeezing the tuber slightly, pearly drops of liquid ooze from the vascular ring. These consist of multitudes of bacteria in a slimy liquid.

From the vascular ring, extension ultimately occurs to the rest of the tuber, which breaks down into a rotten pulp. In this stage many other organisms play a part. Hence the tubers from a diseased crop do not keep, and even mildly attacked ones have a bad flavour, so the crop is of little use.

The stem, roots, and tuber-stalks are also invaded by the bacillus. In the stem, the ring of vascular tissue which surrounds the pith is similarly turned brown, and the vessels are filled with the bacterial mass, which plugs them more or less completely and prevents the fulfilment of



FIG. 113. Ring disease of potatoes (*Bacillus solanacearum*) tubers showing early stages of disease. (After Coleman).

their proper function of conveying water to the leaves. That the wilting is not entirely due to this plugging, but also to the poisoning action of a substance secreted by the bacillus, has been shown by experiments described under tobacco.

Pure cultures of the bacillus (which is fully described on p. 335) have been proved to be able to kill healthy plants, when inoculated into the leaves and leaf stalks by needle pricks, in from 2 to 4 weeks. The inoculated leaf droops in a few days, followed by that immediately above or below it; ultimately the brown ring of affected vascular tissue develops in the stem and at times also in the tubers.

There appear to be two distinct methods of infection of the crop. The first is by the planting of diseased tubers. If not too badly attacked to sprout, the bacteria spread from the seed tuber to the shoot. This may happen with tubers from a diseased crop even though they do not show

the brown ring on cutting, from which it appears that the bacteria may be present (probably in small amount) without causing any clear symptoms. Secondary spread takes place from such primarily infected plants, and is facilitated by the water currents when irrigation is practised,



FIG. 114. Ring disease of potatoes (*Bacillus solanacearum*). the plants in pot on right (except one on extreme right) have been inoculated with pure the bacillus. Those in pot on left were not inoculated. (After Coleman).

the disease spreading along the rows more rapidly than across them. The second method of infection is from the soil, when it has become contaminated by having had a diseased crop of potatoes (or probably also tobacco or tomatoes) previously. Infection in this way probably takes place through wounds in the roots or base of the stem, these being very frequent owing to the occurrence in the soil of organisms, such as nematode worms, which attack underground succulent parts. In the United States, it is also stated that insects which prey on the leaves of diseased plants can spread the infection, but Indian observations seem to show that no great amount of disease is started in this manner.

From the above, it is clear that certain measures can be taken which will effectively reduce the disease. Only such potatoes should be used for seed as have been grown on a plot which has been quite free from disease. It is not enough to discard any showing signs of the brown ring, though when cut sets are planted, naturally none with discoloured pith should be used. Fields that have had a diseased crop should not again be planted with potatoes for some years. The exact length of time

required to purify the soil is not known. Probably it is equally unwise to grow tobacco, tomatoes, or brinjals on infected land. If these two measures are taken, the disease is certain to be greatly diminished. Small holdings in the intensively cultivated tracts, and difficulties in fencing, levelling, and the like in the hills, are obstacles to a proper system of rotation, but should not prove insuperable once the benefits are demonstrated and co-operation amongst the villagers developed. There is a great opening for useful work of this sort in potato-growing tracts where the villagers have been organised for co-operation, especially in the supply of clean seed.

To diminish the rot in tubers from a diseased crop, it is recommended to dig them early and dry thoroughly before storing. They should be kept in a cool, dry place, after removing all that look suspicious. If possible they should be used early and they should never be kept for seed. Rotting ceases below 8°C. (46°F.), so that in some of the hill districts it should not be serious.

With regard to cultivation and manuring, little work seems to have been done on the potato in connection with this disease, but the recommendations given under tobacco wilt (p. 337) should be consulted. The natural checks due to the high temperature of the season following the potato harvest are probably in great measure responsible for the comparatively little damage done by this disease in the Indo-Gangetic Plain, and there is reason to believe that their efficacy can be much increased by hot-weather cultivation. Drainage is also important, as the disease is said in the United States to be worse in fields subject to water-logging.

There are few diseases of plants in India, and certainly none of the potato, which can be more readily checked by measures within the reach of any good cultivator and merely dictated by ordinary common-sense, if once the cause of the damage be clearly understood.

**Blight** (*Phytophthora infestans* (Mont.) de B.).—The potato disease known as "blight," "late blight" or "Irish blight," first attracted general attention in Europe in 1845. For some years previous to this, references to potato diseases in several countries can be found: in France, Belgium, and Germany in 1840 and 1841; in Norway, Denmark, and the United States in 1841 and 1842; in Canada and Italy in 1844. In 1842 also there was published at Munich a scientific account of some diseases of the crop, amongst which many think that the characters of the present disease can be traced. Perhaps, however, the first really trustworthy record of its appearance was at Liège in Belgium in the same year. By 1844

it was doubtless firmly established in a number of centres in Europe and North America. In 1845 a terrible wave of epidemic disease swept the potato fields from Norway to Bordeaux and from Poland to Canada, reaching in several countries to the height of a national calamity. It was seen in Jersey at the end of June, in Belgium early in July, in England (Isle of Wight) about the middle of August and by the end of that month scarcely a sound tuber could be found at Covent Garden market. In Ireland it was noticed in the last week of August and rapidly developed in intensity unequalled in any other country. When it is explained that the potato was estimated at the time to be the staple food of 4,500,000 of the people, the magnitude of the disaster in sight can be realised. In 1846 it was worse if anything. In 1847 milder, but bad again in 1848. The great famine which followed the disease in Ireland has left its mark to the present day; the population of eight millions in 1841 had lost a million by death and emigration by the next census in 1851; the impoverished country was unable to support even these reduced numbers and the stream of emigration then started, and which has only recently slackened, has left Ireland with less than one-tenth of the total inhabitants of the British Isles where formerly she claimed nearly one-third.

In 1845 it was already suspected that the disease had reached Europe from America, and it was stated to be well known at Bogota in the Northern Andes. In after years it was found at Quito on several wild allies of the potato. As this is the area from which the cultivated potato has come, it is not unlikely that it was also the home of the parasite. Its spread would be checked by the tropical regions bounding it on all sides for, as will be seen below, the fungus is unable to survive a high temperature for long. With the advent of quick transport by steamships between 1830 and 1840, and perhaps helped by the growing use of ice at sea, the prospects of safely traversing the tropical zone would be materially increased. Whether North America (as seems probable) or Europe was first invaded is not known, but once northern latitudes were reached subsequent dissemination would offer no difficulties. It is significant in connection with the theory of South American origin that the potato crop in the island of St. Helena was ravaged by a disease for several years prior to 1844, which, from the description, was probably this blight. Though statements to the contrary were made at the time, there is no real evidence that Europe was reached prior to 1840, and little that the blight occupied more than a few restricted areas from 1842 to 1844.

Subsequent spread has been so complete that probably every potato-growing country with suitable climate is infected. Australia was long



thought to be immune, but in 1909 the disease was reported in every State of the Commonwealth\* The history of the introduction and spread of the disease in India is given on p. 129 above

The first sign of disease above ground is the appearance of small, brown patches on the leaves, which, in suitable weather, increase rapidly



FIG. 115. Potato blight (*Phytophthora infestans*) appearance of diseased leaves and tubers.

\* The earlier record, according to which the disease was seen in New South Wales as far back as 1846, is not convincing. Once it has appeared in a particular area it seems to persist unless the climate is so hot that it cannot survive; this is not the case in Australia as a whole, though it would readily account for the disappearance of the disease from particular localities for a time.

in size so as often to involve the whole surface. Extension to the stalk quickly occurs in bad cases, and the entire crown may fall over in a rotten pulp in a day or two. The influence of the weather is most marked. In dry, clear weather, successful infections are limited in number and the resulting spots remain small, brown, and dry, while the stems may escape altogether. In warm, muggy weather, with few periods of sunshine, and ground fogs or heavy dews, the colour rapidly changes to black, the rot is wet, the stems are quickly attacked, and a pronounced smell of decaying vegetable matter is given off and becomes one of the most marked features of the disease. The wet rot and smell are due in great part to secondary organisms, especially bacteria (butyric and others), which rapidly invade the killed tissues.

On the under surface of the leaves (occasionally also on the upper) the fungus forms a whitish haze, consisting of the fructifications emerging from the interior of the leaf. This growth is scanty or even absent in dry weather, and dense but often evanescent on moist, cloudy days.

The underground parts, especially the tubers, are also affected. Sometimes the attack is confined to them, and cases have been described in Australia where there were no decisive symptoms above ground but the tubers were found well infected. It is said that in the great majority of Australian cases it is in the tubers that the disease has been detected, though in especially wet, muggy weather, the above-ground parts are characteristically involved as well. The effect on the tubers (apart from the natural secondary effects of diminution in size and number when the green parts are early damaged) is a dry rot, which does not soften the tissues but causes rusty brown markings just below the skin and extending inwards for a variable distance in an irregular fashion, while the surface of the tuber is marked by corresponding depressions, due to collapse of the underlying cells. In dry soils, nothing more than this may result, and with proper methods of storage, slightly infected tubers will remain unaltered until next planting season, when they may give sound or diseased plants according to circumstances. In damp, heavy soils, however, a wet rot is frequently set up, due to the secondary action of putrefying bacteria and the like, and many of the tubers decay completely before harvest. Both these forms of rot may spread from diseased to sound tubers and cause losses during storage, but the wet rot is the more frequent and destructive, as the parasite fructifies on the surface of the tuber most readily in damp air, and the organisms of wet rot which follow seem also to be more active under humid conditions.

The mycelium consists of branched, hyaline hyphae, 4 to 8 $\mu$  in diameter but varying from place to place according to the size of the intercellular spaces they occupy. There is no regular septation but, as in most allied fungi, the old parts of the mycelium

and also the base of the conidiophore after the sporangia form, often have irregularly developed and spaced cross walls. Only haustoria enter the cells. In the tubers, they are common and easily seen, simple or branched, finger-shaped, clavate, or roundish, and often surrounded by a cellulose sheath, formed by the cell protoplasm in an attempt to shut off the parasite. In the leaves, the haustoria are much harder to detect, filiform, and seldom provided with a sheath, while in the stem they seem to resemble those of the tuber. The walls of cells in contact with, or near, the hyphae turn brown and change in chemical composition, the protoplasm is in part turned brown, in part destroyed, and the starch is slowly dissolved and replaced by sugar.

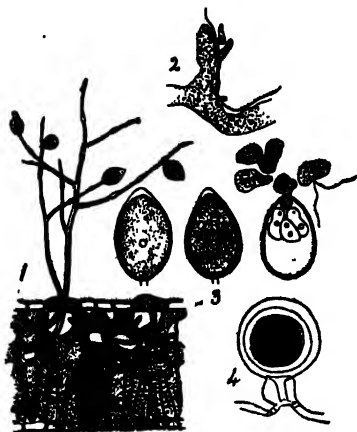


FIG. 116. *Phytophthora infestans*: 1, section of leaf showing mycelium and conidiophores; 2, haustoria in leaf,  $\times 775$ ; 3, sporangia before and during germination showing formation of zoospores; 4, oospore. (1 and 3 after Ward, 4, after Pethybridge and Murphy).

the epidermal cells. The stalk is rather slender (about 10 $\mu$ ), not rigid, and branches above into 2 to 4 branches of variable length (up to 1 mm.) Sporangia are formed at the tips of the branches while they are still short, but growth continues just below the spore, which is pushed over to one side and usually falls off. At each point where growth has thus been renewed there is a little nodular swelling in the stalk, and there may be 9 or 10 such swellings on a branch.

The sporangia are ovoid or lemon-shaped, colourless, and measure from 22 to 32 by 16 to 24 $\mu$ . When sown in water, the apex becomes papillate and zoospores are usually produced. The contents of the sporangium segment into as many polyhedral, uninucleate masses of protoplasm as there are to be zoospores, each provided with a central vacuole. The papilla then bursts and the zoospores emerge fully formed, though sometimes two or more may adhere for a time. After a few seconds' rest in front of the opening, they swim off by means of two motile, oar-like cilia, and in from a quarter of an hour (at high temperatures) to 24 hours (at low temperatures) settle down, become quite round, surround themselves with a cellulose wall, and soon germinate by a germ-tube. Many spores may germinate directly by a germ-tube from the apex, especially such as are old, not fresh, not immersed in water, exposed to high temperatures, or sown in organic solutions; but the normal method is by zoospores. The germ-tube may soon bear a secondary sporangium or may infect a suitable host and give a parasitic mycelium. The spores are disseminated by wind, water, and possibly insects or other animals.

Oospores were long unknown, but have been found several times during the past few years as a result of improved methods of artificial culture. They have not yet been

found in nature. Unlike the similar bodies previously known, the antheridium develops before the oogonium, the latter arising on a hypha which penetrates the antheridial cell from side to side. It swells up as it emerges, so that the antheridium surrounds the stalk of the oogonium like a collar. Fertilisation occurs doubtless, as in the allied *Ph. erythro-septica*, by the passage of a nucleus from the antheridium into the base of the oogonium. The oospore averages about  $30\mu$  in diameter and lies loosely in the oogonium, which is pear-shaped or almost spherical and  $31$  to  $46\mu$  across. The oogonial wall is smooth and reddish-brown, that of the oospores also smooth but colourless and up to  $4\mu$  thick. In many cases, no antheridium is formed, the oospore then developing parthenogenetically. Germination has not been observed.

In artificial cultures, too, it is not uncommon to find certain cells cut off, usually at the tips of hyphae, which are probably of the same nature as the chlamydospores found in allied species (see p. 14). They are irregular in shape, often roughly pyriform,  $24$  to  $38\mu$  in diameter, with a thick, pale amber-coloured wall, and dense, oily contents. They have not been observed to give new hyphae.

Infection occurs through any part of the epidermis of the leaves and stem, not merely through the stomata. Even the corky rind of the tubers can be penetrated by the germ-tubes, especially in immature tubers. The under surface of the leaves is more readily attacked than the upper. New fructifications may appear in 4 or 5 days on the leaves and in 16 days on the tubers, but are not common on the latter except in very damp air or when the rind is abraded. Besides direct infection by means of spores shed on the soil and washed down into contact with the tubers, some hold that the mycelium may travel down inside the stem to the underground parts; in other cases it is said that the tubers are infected by spread from the seed-tuber, even when the above-ground part remain healthy. But it is certain that the infection usually occurs from spores shed from the leaves.

Temperature has a strong influence on germination and infection, the best temperature for both being between  $10^{\circ}$  and  $13^{\circ}\text{C}$ , though the mycelium, germ-tubes, and conidiophores grow best about  $22^{\circ}$  to  $25^{\circ}\text{C}$ . Germination by zoospores ceases below  $2^{\circ}\text{C}$  and above  $23^{\circ}$  to  $26^{\circ}\text{C}$ . Germination by a germ-tube is scanty below  $10^{\circ}\text{C}$ , most frequent about  $23^{\circ}\text{C}$ , and ceases near  $30^{\circ}\text{C}$ . The living mycelium in the tuber can be killed by exposure to  $40^{\circ}\text{C}$ . dry heat for 4 hours or  $30^{\circ}\text{C}$ . for 65 hours. This is from European investigations but in Australia it was found that 4 hours at  $49^{\circ}\text{C}$ . ( $120^{\circ}\text{F}$ .) were required. The tubers are not injured for seed until the temperature exceeds  $130^{\circ}\text{F}$ . In moist heat in Australia, sporangia developed at  $31^{\circ}\text{C}$ , but in dry heat, none were formed above  $27^{\circ}\text{C}$ . It is possible that these figures would not be strictly correct for India, but it has been found at Pusa that neither the mycelium in the tubers nor the fungus in pure culture can long survive the temperature of the laboratory in the hot weather, when it ranges from  $30^{\circ}$  to  $35^{\circ}\text{C}$ . Hence there is little fear of the disease becoming endemic in the plains. Neither light nor oxygen seems to influence germination.

As the sporangia only retain their vitality for about 3 weeks (much less if dried), and as resting spores have not been found in nature, the persistence of the disease from year to year requires explanation. It cannot be said that an entirely satisfactory picture of the manner in which the yearly attack originates has been offered; but it is conclusively established that the mycelium in the tubers survives in many cases without causing great damage, and on planting is able to renew its active growth and may pass into the tissues of the young shoot, growing with it and fructifying on its surface above ground, if the weather conditions

are suitable. At the same time, it is well known that diseased tubers give quite sound plants or else fail to sprout altogether, in the majority of cases, and so various additional explanations have been offered. It is possible that the mycelium survives in the soil or in rotting debris of the plant until the next season, especially if the thick-walled cells above described (from cultures) are formed in nature; oospores may also occur and have merely been overlooked (though the search has been long and thorough). It has been stated more than once that experience has shown that potatoes grown in soil which bore a diseased crop the previous year suffer more than others, yet attempts to furnish exact experimental proof of soil infection have failed. All that has been established is that tubers from the old crop may bear spores, if exposed to the air and kept moist the following year. It seems difficult to avoid the conclusion that the disease is sometimes started in this way. The chief difficulty in accepting these explanations as sufficient is one already discussed in a similar connection in wheat rust; the disease does not usually appear until the plants are from 2 to 4 months old, indeed it is rare until they have finished flowering, whereas if the above views were correct we would expect it in the young plants. The factors here concerned have probably to do with variation in the degree of resistance of the host at different periods of its growth, or the influence of external conditions, such as temperature and moisture, on the development of the fungus.

The chief predisposing causes are excessive humidity of the air and a suitable temperature. The very moist climate of much of Ireland has established that country as one of the heaviest sufferers. In 1879, for instance, the loss was estimated at £6,000,000. In the United States, the endemic area is in the north-east only, the rest of the country being either too dry or too hot for it. In Australia, late planting in certain districts has enabled the moist season to be avoided and the disease has consequently been reduced. In South Africa, it was severe near Cape Town in October 1909, but it disappeared as if by magic when the dry south-east wind set in in November. In India, the connection with temperature and moisture of the two Bengal outbreaks has already been discussed (p. 129).

Where the mean temperature exceeds 77°F., the disease is said to be unknown. It has frequently been observed that epidemic outbreaks are likely to occur when unusually cool weather sets in at the critical period. A fall to below 60°F. is very liable to start an attack. This is due to increased germination of the sporangia and not to any increased

susceptibility of the plant. After infection, the attack develops most rapidly if the temperature rises towards the optimum for mycelial growth.

Over-manuring has been frequently blamed for severe attacks. This effect appears to be confined to nitrogenous manures such as farm-yard manure, phosphates and potash rather increasing the resistance of the tubers. Over-irrigation was blamed in Bengal in 1902, very possibly with justice as in some places water was given every 6 days. In South Australia also, irrigation, once the disease has started, is said to intensify the tuber rot.

In the Khasi and Nilgiri hills, two crops are usually got in the year, the second being largely for seed. The same land is often used year after year in these tracts and in Bengal. If soil infection occurs, it is obvious that such practices must favour the disease.

The advantage of early or late sowing depends entirely on the local meteorological conditions. In 1902, the late-sown potatoes were most damaged in Bengal, in 1912, they almost escaped. This was apparently connected with the January-February temperature and humidity.

Since tuber infection takes place largely from spores shed on the soil from the leaves, tubers near the surface are most frequently attacked. Hence covering them by deep ridging, shortly before flowering time, has been advocated. The soil is heaped up round the base of the plants to a depth of 4 or 5 inches. The practice undoubtedly lessens the tuber rot but it has been found not to pay, as the yield is diminished. Since this is probably due to increased loss of water by evaporation from the ridges there would be less objection to it under irrigation, than where the crop is rain-fed. With the heavy rainfall of some of the hill districts it might also be profitable.

Since the tubers can be infected at any age and whether the skin is sound or abraded (though more readily in the latter case) there is obvious danger of infection occurring while the crop is being lifted, if diseased tops, bearing spores, are still present. Hence early digging is often unwise. In New England it is recommended not to dig until a week after the death of the tops, except on low, heavy soil in exceptionally wet seasons, when tuber rot spreads rapidly and early digging is necessary. In Ireland, farmers are advised to dig as soon as the tops have no green left.

Losses in storage are not likely to occur in the plains from this fungus, as the high temperature kills it in the hot weather. In the hills there is no definite information, but as the storage seasons are reversed,

the low temperature of many hill tracts may check spread. A medium high temperature is the worst; thus in America tubers from a diseased crop lost 79 per cent. after two months storage at 70°F., 53 per cent. at 55°F.; and only 17 per cent. at 40°F. Drying the tubers before storage is advantageous and so is a free circulation of air in store. The use of lime and disinfection with formalin have proved useless.

It is clear from experience in the two Bengal outbreaks that if hill or other infected seed be used in the plains, it should be got in fairly early and exposed for a month or so to the heat of the late rains. If for any reason this cannot be done, it is better to use seed from Bombay or some other disease-free area.

Spraying to check potato blight is probably more widely employed and gives more satisfactory results than with any other field crop disease. Not only is it a routine practice in the intensive cultivation of such favoured places as the Island of Jersey, where high profits permit of lavish expenditure, but in the poverty-stricken districts of the west of Ireland nearly 10,000 sprayers were supplied by the Agricultural Department to the potato growers in the 6 years ending 1914. At the Vermont Station in the United States, the average gain for the 20 years ending 1910 was 64 per cent. (105 bushels per acre) above the non-sprayed plots. Part of this was due to checking other pests. The average gain in New York State from thorough spraying (5 to 7 times) is calculated as a result of 10 years' extensive experiments to be somewhere between 50 and 100 bushels. Under farm conditions the profit was about Rs. 60 per acre. These were with Bordeaux mixture. In Ireland, with simpler methods and less frequent sprayings, the average increase from 6 years experiments was 1 ton, 14 cwt. (nearly 50 maunds) with Bordeaux mixture and 2 tons, 10 cwt. (70 maunds) with Burgundy mixture. The only records in India are from the Khasi Hills, where the frequent rain makes the operation less certain. On a small scale in 1906 the increased yield from plain Bordeaux mixture was at the rate of 50 maunds per acre, yielding a profit over the cost of spraying of nearly Rs. 50, while with resin-Bordeaux the increase was 64 maunds and the profit over Rs. 60.

Spraying should commence before the disease ordinarily appears, a period which can only be determined by experience in each locality. In the United States, it is begun when the plants are 6 to 8 inches high and is repeated every 10 days or fortnight, as many as 6 sprayings being often advised. In Ireland, they begin about June and spray again in 2 or 3 weeks, giving a third spraying later on in wet seasons.

Burgundy mixture seems to be preferable to Bordeaux in many places, and should be used in India if there is any doubt about getting good lime. The instructions for its manufacture are given on p. 143.

The quantity required per acre for each spraying of an average crop in Ireland is about 100 gallons, but with less foliage (as usual in India) a smaller amount will do. The cost varies according to the price of the materials, but has always proved slight in comparison to the gain, in localities where blight is prevalent, and ought not to reach 25 per cent. of the added profit.

The variation in susceptibility to blight shown by different kinds of potatoes has long been known (see p. 122). The production of disease-resistant varieties is a specialised art and, as the successful sorts rapidly become available to growers, need not concern us. The experience with English potatoes in the Khasi Hills is not, however, quite satisfactory. Resistant varieties have been found to maintain their superiority for a few years only, after which they succumb as readily to blight as any others.

The parasite occurs on a number of other Solanaceous plants, the only one of economic importance being the tomato. The disease in this crop is sometimes of equal severity to that in the potato, which it resembles in all important respects.

Several other species of *Phytophthora* are known to be able to grow on the potato, but only one has been found to cause a disease of the crop under natural conditions. This is *Phytophthora erythroseptica* Pethybr. which is said to be closely allied to the fungus which causes the "kole-roga" disease of the Areca palm in South India. It has not been found attacking the potato crop in India.

**Leaf blotch** (*Cercospora concors* (Casp.) Sacc.).—This leaf disease is not uncommonly confused with blight (*Phytophthora*) but is far less injurious in its effects. It was described in Germany many years ago, and has recently been found in several other parts of Europe and also in the United States. In India, it has been seen in Bengal, Bihar, and Bombay and is of regular occurrence in places (as at Pusa) where *Phytophthora* is not known.

The attack causes light green, roundish spots to appear on the leaves, at first usually on those near the ground. The spots are not sharply limited and gradually turn greenish-yellow, yellow, and finally brown. They are up to  $\frac{1}{2}$  inch across when fully developed, but may coalesce into



larger patches. Their under surface is ultimately covered with a grey-violet growth, the fructifying stage of the parasite.

The hyphae are intercellular, colourless, and with rather scattered septa at first, then turn grey-brown and form short, barrel-shaped or roundish cells with oily contents. It is probable that this last form is a more durable condition, capable perhaps of living over in the soil, after the leaves disintegrate, until the new crop appears.

The conidiophores arise chiefly from the internal mycelium, emerging in clusters through the stomata of the under surface of the leaf, less often above. They are grey, sparingly septate, 40 to 75 $\mu$  long by 7 $\mu$  broad, and sometimes branched, the branches being short, curved, and mostly from one side. As in most other members of this genus and of such allied forms as *Helminthosporium*, the first spore is terminal, but growth continues from below its insertion just as in *Phytophthora*, the position of the earlier spores being marked by an abrupt bend or "knee." Hence the upper part of the conidiophore has a gnarled or knotty appearance. Sterile hyphae also come through the stomata and give rise to a superficial mycelium on which erect conidiophores may be borne.

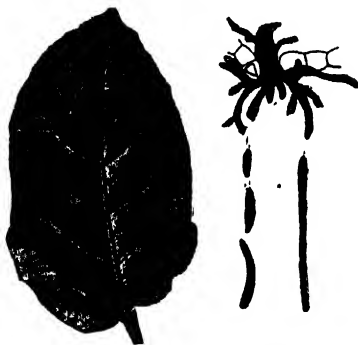


FIG. 117. Leaf blotch of potato (*Cercospora con-cors*): leaf with blotches,  $\times 8$ ; conidiophores and conidia (after Lagerheim and Wagner).

The conidia are colourless or very faint grey, elongated, straight, or slightly curved, narrowed above but expanding again slightly just at the tip, and divided by 1 to 4 (usually 3) septa. They measure 12 to 50 by 5 to 6 $\mu$ .

The affected leaves gradually turn yellow and dry up, with the result that the growth of the tubers is checked and the crop may be materially reduced. The attack occurs at the same time as *Phytophthora*, when the crop is about full-grown and the tubers are forming. In India it has not been observed to do much damage.

The treatment recommended is spraying as for blight. Unless the latter disease were also present it would probably not pay in India. Our climate seems to be too dry, at least in the plains, for the parasite to develop freely. Varieties are said to differ greatly in their liability to this as to other potato diseases, but there are no observations on this point in India.

**Early blight** (*Alternaria Solani* (E. & M.) Jones and Groul).—Unlike the true blight, this disease has gradually increased in severity

in the 25 years or so since it was first considered as of economic importance. It seems to have originated in the United States, as all the earlier records are from there. It is now known in practically every part of the world, from Canada to New Zealand and from Japan to South Africa. In this country it occurs in the Indo-Gangetic Plain and the Nilgiris.



FIG. 118. Early blight of potato (*Alternaria Solani*); leaf (after Galloway); conidia (after Jones and Grout).

The attack usually begins shortly before the tubers commence to form, two or three weeks earlier than the common blight. It continues to develop until harvest, so that both blights may co-exist. Rather small, isolated, pale brown spots appear, scattered irregularly over the leaf, often mostly at the tip and margins. As they grow, they become irregularly circular and show concentric, narrow, darker lines following the outline, which remains sharply defined. These characters distinguish the spots from those caused by *Phytophthora* or *Cercospora*. The older spots are dark brown, dry, and brittle but do not usually break away. Several may unite, but increase in size is slow and general infection, such as occurs with *Phytophthora*, is not found. The intervening parts of the leaf fade and there may be considerable curling from irregular shrinkage of the tissues as they dry up, but death is slow and the whole plant never collapses from wet

rot. Usually, only the older leaves are affected unless the plant is weakly or, attacked by *Phytophthora* or insects, when all may be involved, especially in the neighbourhood of local injuries. In the late stages of severe attacks, the stalks turn yellow and dry up.

The effect on the crop may be considerable, as the attack often coincides with the period when the developing tubers throw a strain on the nutrient resources of the plant. The tubers are not directly attacked and are never rotted, but they remain small, immature, soft-skinned, difficult to keep, and deficient in starch. Losses of 50 per cent. have been recorded in America, but in India the damage is generally much less.

The mycelium consists of light brown or greenish hyphæ, running in and between the cells at the spots. Conidiophores emerge through the stomata or between the killed epidermal cells, and are scattered over the dead centre of the spots, not the living margins. They are about 50 to 90 $\mu$  long by 8 to 9 $\mu$  broad, septate, somewhat curved, and enlarged at the tip to a cup-shaped head on which a single spore is borne, rarely two near together. The conidia are very variable in shape but mostly very long bottle-shaped, the neck much attenuated and sometimes half the length of the whole spore, and the base slightly narrowed. They measure 145 to 370 by 16 to 18 $\mu$  and are divided by 5 to 10 transverse septa, some of the broader compartments being sometimes further divided by a longitudinal wall. The lower part is brown, the neck almost colourless. In very vigorous cultures, chains of two (perhaps more) spores are sometimes found, but not on the leaf where spore production is usually scanty. A form with almost cubical spores also occurs in England but further work is necessary to establish its identity with the true *Alternaria Solani*.

Germination can take place from any cell of the spore, the germ-tube entering the leaf both through the stomata and directly into the epidermal cells. New spots appear in from 3 to 6 days. If the leaf is kept very moist or has been injured by Phytophthora or leaf-eating insects, infection occurs more readily. Old bits of potato leaf infested with the mycelium will give a new mycelial growth even after a year and cultures 8 months old have been found still alive; the spores also will germinate when a year old though not after two years. Hence the disease is readily perpetuated from season to season. Of recent years, some English mycologists have given evidence that the mycelium reaches the tubers and hibernates in them, just as with Phytophthora, though there is not any such need of this special way of surviving, when the crop is off the land, as in the latter case.

The disease is not dependent on atmospheric humidity to anything like the extent blight is. It spreads rapidly under relatively dry conditions and is only checked by severe drought. It is said to be much in evidence in Australia on light, sandy soils. Apart from this, anything that tends to lower the vitality of the crop favours the disease, as the fungus (unlike Phytophthora) belongs to the group of "weak" parasites (of low virulence) which do little damage to plants in their fullest vigour. It is claimed in New Zealand that growth for several generations on leaves killed by Phytophthora entirely destroys the virulence of the fungus, but it is not certain that the experiments were not partly carried out with the saprophytic allied species, mentioned below, which commonly occurs on the same spots.

Spraying has yielded excellent results in the United States, where alone, at present, active measures seem to be called for. The procedure is the same as against Phytophthora but the first application should be two or three weeks earlier.

There is great danger in neglecting rotation where this disease is prevalent, owing to the pronounced vitality of the mycelium and spores. Potatoes are often grown year after year in the same land in Bengal, and even two crops a year in the Khasi and Nilgiri hills. It has been observed

in similar cases that the disease gradually increases. Clean removal and burning of infected tops at harvest is strongly recommended for the same reason. As tomatoes are also attacked, they should not follow potatoes in infected areas. If the mycelium hibernates in the tubers, as stated in England, clean seed from a disease-free crop should be used. Some varieties are said to be less damaged than others and their use might be extended.

As with all other parasites that prefer weakened plants, high cultivation, heavy manuring, and anything that stimulates the crop, lessens its liability to attack.

In several accounts of this disease, a second species of *Alternaria* (*A. fasciculata* (C. & E.) Jones and Grout), which commonly co-exists with *A. Solani* on the spots, has been confused with the latter. It can be readily distinguished by the fact that its spores frequently occur in longish chains on the leaf, *A. Solani* only rarely forming short chains in culture. *A. fasciculata* is saprophytic and seems to occur frequently on dead or dying plant tissues, having received a number of different names on different plants. Both forms seem to occur also on the tomato and to have been indiscriminately referred to under the name *Macrosporium Tomato* Cke., a fungus to which one of the tomato fruit rots has been attributed. No successful attempts to produce this disease have been recorded, while there have been many failures, due possibly to using the saprophyte for the inoculations. At the same time, *A. Solani* has been proved to attack the leaves and stem of the tomato and it seems likely that it can also attack the fruit.

The fungus is also found on some other allied plants, as *Datura* (*Datura Stramonium*), *Hyoscyamus niger*, *Solanum nigrum*, and *Solanum Commersoni*.

**Root and tuber rot** (*Hypochnus Solani* Pril. & Del., *Rhizoctonia destruens* Tassi, and *R. sp.*).—The potato is attacked by the same two species of *Rhizoctonia* as have already been fully described under cowpea (p. 262). A third distinct form, which has been identified as *R. destruens*, causes a disease of the growing crop at Poona and a rot of stored tubers in Bihar.

*Rhizoctonia destruens* attacks the growing crop at and below the ground level, the parts becoming covered by a web of hyphæ with sclerotia. The tissues are rotted and the leaves dry and turn brown. The tubers usually appear sound, though reduced in size; but cases have

been observed of infection without outward signs through the attachment to the parent stem, and this doubtless accounts for the storage rot. In the latter, the tubers are softened and have thick, white strands of mycelium and brown sclerotia on the surface.

The hyphæ are like those of the other species but have a tendency to unite in strands. Clamp-connections are numerous. The sclerotia are light brown on the surface, white inside, with a distinct cortex, round or nearly so, and 1 to 2 mm. in diameter. They are sometimes stalked and occasionally united in clumps. No perfect stage is known.

Inoculations on tubers succeeded always if the tubers were wounded and sometimes also through unwounded eyes and lenticels.

The parasite attacks wheat, lentils, groundnut, lucerne, betel pepper, Delphinium, and several other plants. There is no evidence of specialisation on the different hosts.

The treatment has already been discussed (p. 266). The tuber rot can probably be reduced by dipping the tubers into corrosive sublimate solution of 1 part in 1,000 parts of water, or formalin 1 per cent. The former is only safe for seed potatoes, making the tubers unfit for food.

## THE BRASSICAS AND ALLIED PLANTS.

CABBAGE (*Brassica oleracea* L.)

CAULIFLOWER (*Brassica oleracea* L. var. *botrytis*).

TURNIP (*Brassica campestris* L.).

SARSON (*Brassica campestris* L. var. *Sarson* Prain).

TORIA (*Brassica Napus* L. var. *dichotoma* Prain).

RAI (*Brassica juncea* Coss.).

BLACK MUSTARD (*Brassica nigra* Koch).

CABBAGE-LEAVED MUSTARD (*Brassica rugosa* Prain).

DUAN (*Eruca sativa* Mill.).

RADISH (*Raphanus sativus* L.).

**White rust** (*Cystopus candidus* (Pers.) Lév.).—This disease is found on a number of cruciferous plants throughout the world, its hosts including the turnip, cabbage, cauliflower, radish, horse-radish, mustard, cress, rape, and many weeds. In India, it has been found on turnip, radish, and duan, and it is probable that this list will be extended on further observation. It is not confined to the Cruciferae, occurring on Cleome (Capparidaceae) in India and on other members of this order elsewhere.

All parts of the plant may show the symptoms of attack except the roots, the parasite appearing on the surface of the leaves, stem, and inflorescence as prominent white pustules, very variable in size and shape and often confluent. Marked swelling and distortion of the attacked parts often results, especially in the inflorescence, as in the radish. In these cases the axis of the inflorescence, and the flower stalks, may be

enormously thickened, up to 12 or 15 times the normal diameter, while the floral organs become wholly or in part swollen, fleshy, green or violet in colour, and persist instead of the petals and stamens falling early. The



FIG. 119. White rust (*Cystopus candidus*) and downy mildew (*Peronospora parasitica*) of *Eruca sativa*, showing hypertrophy of stem and flower,  $\times \frac{1}{4}$ .

petals may become like sepals, and the stamens leaf-like or occasionally like the carpels (seed leaves). The latter may be open, while the ovules are usually atrophied, as are also the pollen grains and thus sterility is caused. The pustules of the parasite are usually developed on the swollen parts; when absent, a second type of reproductive body, the oospores, is generally found hidden within the swelling.

The leaves are not often greatly modified. On some hosts, however, those found on totally infected stems may be thickened, fleshy, pallid, and distorted or inrolled. If the infection is partial

or not intense, there are usually only scattered pustules on the under surface or, less often, on both sides. Sometimes the leaves are reduced in size over all or a part of the plant. In these general infections, the whole plant may be dwarfed and form no flowers or only imperfect ones.

The stem swellings may be confined to a small area, sometimes on one side only, the other being unaltered. In other cases, the whole stem is uniformly swollen for a length of several inches. Sharp bends, sometimes angular, sometimes in a spiral, are often found at the swollen areas

In many infected stems, the small buds in the axils of the leaves which normally do not sprout, grow out into lateral branches some inches in length.

In the flower stalks, the same general type of hypertrophy occurs. When the stalk is much swollen the flowers may be entirely arrested in their development, or all the floral organs may themselves be hypertrophied in a regular manner. Not infrequently the stem and flower-stalks are unaltered and only the flowers are attacked.

In the flowers, sometimes only a single sepal is hypertrophied, sometimes only the ovary, but usually all the organs are swollen and turned green or violet. Occasionally they throw back to the spiral arrangement found in more primitive plants. The sepals may be immensely increased in size and thickness, often deeply concave on the inner surface, and sometimes provided with cylindrical or flattened appendages near the base. The petals are also enlarged, vary much in shape, and sometimes have nopaloid characters. The stamens are usually greatly altered being thickened into a club-shaped body in which the anthers are represented by an oblong, grooved, green mass, or transformed wholly, or the anthers only, into small, leaf-like structures. In some hosts, the stamens are less affected and may even bear pollen in a portion of the pollen-sac, the rest being sterile; occasionally supplementary pollen sacs are formed. Of more interest is the appearance of stigma-like structures at the tip and rudimentary ovules on the margins of certain altered stamens, which thus come to have the characters of carpels. The pistil itself may be enormously swollen into a conical, thick-walled sac, or transformed into a couple of carpellary leaves, borne on a common stalk. In the former case ovules may be formed, but they remain sterile and dehiscence of the fruit does not occur.

The internal effects of the parasite are of interest. In hypertrophied leaves, the differentiation of the leaf-parenchyma into palisade and spongy tissue is lost, all the cells of the mesophyll being alike and twice or three times the ordinary size. In thickened stems, the increase is due to a modification of the cortex into a tissue of large, thin-walled cells with few intercellular spaces, sometimes formed merely by the enlargement of the existing layers, sometimes by the development of new layers through division of the cortical cells. In some hosts (cress) the cells of the endoderm and pith also multiply by division. The thick-walled sclerenchyma between and over the bundles is altered to a thin-walled parenchyma like that of the cortex. The cambium of the bundles retains its activity longer than the normal and the new elements are somewhat longer; radial bands of parenchyma also occur in the xylem. In those bundles found in the sprouting lateral shoots, it has been observed that only spiral and a few pitted vessels are found, with little or no lignified tissue between, and a general absence of secondary thickening of the cells. Between the bundles, an interfascicular cambium may also continue to form new parenchymatous elements. The epidermis increases both as regards size and

number of its cells, and the number of stomata is augmented. In the thin-walled parenchyma, especially in the outer layers, chlorophyll and starch are copious and, in addition, there is often a reddish-violet pigment in the cell sap in the neighbourhood of the conidial beds and the oogonia. Starch may be also found in the cambium, endoderm, pith, and other parts in which it is usually absent. In the branches of the inflorescence, the same general changes are found.

The mass of the hypertrophied parts of the flower consists of parenchymatous tissue like that already described in the infected stems. The cells are very much enlarged and the number of layers is increased, intercellular spaces are few or absent, and chlorophyll and starch usually copious, even in the petals and stamens. The nucleus of these cells is very large and occupies the middle instead of lying against one of the walls. It shows a tendency to become lobed and even to divide into several fragments, each provided with a nucleolus. In all the organs, the number of fibro-vascular bundles may be increased by the development of numerous accessory bundles, and the individual bundles may also be enlarged by the prolonged activity of the cambium. The new tissue consists chiefly of vascular elements and radial bands of parenchyma, the fibrous part being reduced. Special cells containing an aromatic substance known as myresine are often found in abundance, whereas in normal plants they are sparse. The pollen sacs and the spiral cells of the anthers may be replaced by chlorophyllous parenchyma. The epidermis is altered, losing the differences which are observable in healthy plants between the epidermal cells of the two surfaces. Stomata become more numerous. Both the number and size of the cells may be increased; sometimes they contain chlorophyll, absent in normal plants; so also hairs may develop where ordinarily there are none, as in the anthers; and stomata may appear in the petals, which may further lose their epidermal papillae. Even more remarkable is the change in the inner epidermis of the swollen ovary or fruit, which becomes transformed from a layer of very long, narrow cells, with walls of equal thickness all round and without stomata, to a layer of large, irregularly polyhedral cells, only the outer walls of which are thickened, and which is broken by numerous stomata. This layer seems to serve as a water-storing tissue.

It will be seen that the tendency throughout is to a lessening of the differentiation of tissues, particularly such as are secondary. Further, there is a similar tendency to reduce the differentiation of organs. Thus petals come to resemble sepals; petals, stamens and carpels show leaf-like characters; and stamens become like carpels even to the extent of being provided with a sort of stigma at the tip and rudimentary ovules on the margins. Internally, the general result is the development of a nutrient tissue of a more or less constant type, resembling that already described in the maize galls caused by *Ustilago Zeæ*, in all those parts of the host plant where the parasite is about to form its fructifications. This tissue is rich in starch, partly formed *in situ* through the agency of the chlorophyll with which the nutrient cells are often provided, partly carried in from other parts of the plant, as is suggested by the increase in the vascular system. Up to a certain point the accumulation of food supplies in the nutrient tissue is more than sufficient to counterbalance their consumption by the parasite. Later on, however, usually when spore-production starts, the parasite consumes more than the host can manufacture. The result is the depletion of the nutrient cells and their consequent collapse and death. This is marked by the drying up and turning brown of the infected parts and sometimes by complete disintegration of the soft tissues.

The hyphae are unseptate and spread exclusively between the cells of the parenchyma, especially that modified as already described in the hypertrophied organs but also in other parts of the plant, even those that seem outwardly sound and unaltered. Numerous very small, spherical or knob-shaped haustoria are sent into the nutrient cells, often a large number into each cell. These haustoria appear to remain functional for a short time only, and then degenerate. They do not disappear after death but merely lose their protoplasm and usually become covered by a layer of cellulose, which forms a circular



coat entirely cutting the haustorium off from the cell protoplasm. This layer is formed by the activity of the protoplasm, in the same manner as the sheaths found enclosing the infection hyphae of *Ustilago Zeae* in maize. The haustorium itself, after penetrating the cell wall, does not grow into the cell for any distance, nor does it rupture the protoplasmic layer lying on the inside of the wall, merely pushing the latter before it into a concave depression. Until a late stage in development, the cell does not suffer. Its activities are even, in many cases, stimulated by the influence of the parasite, as is indicated by the increase in size, division into new cells, and formation of chlorophyll and starch where usually there is none.

Branches from the internal mycelium now collect below the cells of the epidermis, into which quantities of haustoria are sent. From these hyphae, which remain distinct and do not form a pseudo-parenchymatous stroma as in the rusts, the sporangial beds arise. These are formed by the vertical growth of broad, short, club-shaped sporangiophores under the epidermis, which is raised up into a blister, separated from the cells below by the layer of sporangiophores. The latter are free from each other laterally and are very thick-walled, especially towards the base. They measure about 35 to 40 by 15 to 17 $\mu$ . The epidermal cells next undergo a change which results in an increase in size, accompanied by great thickening and gelatinization of the wall all round, and softening of the middle lamella separating the cells. When moist weather follows, they swell and rupture along the softened middle lamella, exposing the sporangial bed.



FIG. 120. *Cystopus candidus*: sporangia and oospores. (After Berlese).

The sporangia are formed in basipetal chains, the first-formed being closely pressed against the still unruptured epidermis. This spore is not capable of germination, and its apex, which is rather closely united with the inner wall of the epidermis, is provided with a cellulose thickening. Its function is probably to aid in the alteration of the epidermis which ultimately results in the rupture of the latter. The chains of sporangia thus exposed to the outer air break up into their individual elements. This occurs by the solution of the jointed neck between each pair of sporangia, which is formed of a disk of a substance known as callose, capable of undergoing a chemical modification which renders it easily soluble in water. In dry weather the loosened sporangia are disseminated by currents of air. They are round, hyaline, with uniform thin walls, and measure 12 to 18 $\mu$  in diameter.

The germination of the sporangia occurs if they are completely immersed in water while fresh, and is limited to about six weeks after formation. The apex becomes drawn out to a rounded papilla, and a few large, irregular vacuoles appear in the protoplasm. After a time, these become quite spherical and discharge to the outside. Then the whole contents segment into as many polyhedral fragments as there are to be zoospores. Each of these is provided with a contractile vacuole. The zoospores, of which there may be from 4 to 8, separate from one another and escape to the outside one by one, or sometimes in adherent groups, or the whole mass may be emptied into a sort of bladder formed by the swelling of the beak, as occurs in *Pythium*. They remain in front of the opening for a few minutes, during which the cilia appear and movement begins. They then swim off in all directions. They are of the usual Peronosporaceous type, flattened ovate to kidney-shaped and with two cilia from the flat or concave side. They come to rest after a time, surround themselves with a cell wall, and germinate by a germ-tube, which is capable of entering through the stomata of the cotyledons, leaves, and probably young inflorescence and developing into a parasitic mycelium within suitable host plants. The germination of the sporangia is closely dependent on the temperature. High temperatures

are unsuitable, the maximum being about 25°C. Low temperatures both increase the rate of germination and cause a greater number of sporangia to liberate zoospores. About 10°C. is the best. The time required varies from about 2 to 10 hours, but occasionally is only 45 minutes. Light seems to have little effect.

Oospores are formed in abundance in some hosts and in some localities, in others they are rarely found. They can generally be most easily obtained from hypertrophied parts of the stem and inflorescence, occupying the nutrient tissue sometimes in great numbers. They are not uncommon in the leaves of some hosts, as in the common weed *Cleome viscosa* in Dehra Dun. They are always formed inside the host, and the swollen parts containing them may bear no sporangial beds; this is particularly noticeable in cress and turnip. In the latter plant, cases have been recorded where search in many individuals has revealed oospores only in the swollen pistil in a few cases and these bore no sporangia.

The oogonia are roughly spherical, about 50 to 60  $\mu$  in diameter, and with a single clavate and much smaller antheridium. After fertilisation, a wall appears, cutting off the outer zone of protoplasm from the rest of the oogonial contents. The outer part is the periplasm, the inner becomes the oospore. The wall of the oospore thickens, turns brown, and is provided with low, blunt ridges, often branched and sinuous in outline. The mature oospore measures 40 to 55  $\mu$  in diameter. It germinates when immersed in water after a period of rest of several months, long before which it has usually been cast into the soil. The exospore ruptures by an irregular crack and the endospore is pushed out into a thin bladder into which the contents of the spore pass, having first segmented into a number (up to 100 or more) of polyhedral masses, just as in the sporangium. In the bladder they separate, develop two cilia, and begin to move in an irregular, jerky fashion. Soon the bladder wall dissolves and the zoospores swim off in all directions. They are exactly like those from the sporangia in character and ultimate behaviour, and there is no doubt that many of the early attacks in annual plants are due to infection by zoospores from germinating oospores left in the soil, or in plant debris from the previous seasons' crop.

It was formerly believed that infection by *Cystopus candidus* is only possible in the majority of cases through the cotyledons (first seed leaves) and that the older leaves and stem do not allow the fungus to enter. More recent experiments show that there is no marked difference in the susceptibility of leaves and cotyledons in several plants. The young tissues of the inflorescence seem to be very readily attacked, and often bear pustules when the vegetative parts below are not infected. Two types of infection, a general and a local, may probably be distinguished. In the first, the whole plant may be stunted and sporangial beds develop all over the leaves, stem, and inflorescence, the fungus spreading through all the plant. In the second type, only a single flower may be attacked; and the existence of the cases noted above where one sepal alone is hypertrophied, probably depends on localised infection of the flower bud. Entry can only take place through the stomata in all cases.

There is still some doubt as to how far the various forms on different hosts are specialised races, similar to those already referred to in the

cereal rusts. The form on radish has been found to be able to infect other species of *Raphanus*, and also white mustard and cabbage less readily ; with the latter host not 1 per cent. of the infections succeeded. On the turnip, cress, and some other hosts, no infection was obtained. Another observer found that the form on turnips infected cabbage and its varieties as well as other plants ; while that on the weed *Arabis alpina* infected 9 other plants belonging to 6 genera, but failed on radish, mustard, and cabbage. As, however, it is often difficult to secure the conditions proper for infection with this fungus, the negative results are not of absolute value. The positive results show that specialisation is not of a very high order.

*Cystopus candidus*, when it occurs alone, does not often do much damage. Frequently, however, it is associated with the downy mildew, *Peronospora parasitica*, and instances are known of considerable injury resulting from the combined attacks of these two fungi. Sometimes white rust has been recorded as causing a good deal of damage in cabbage and its varieties.

Treatment is not usually called for, except when the downy mildew is also present, when the measures recommended below under that head may be taken.

**Downy mildew** (*Peronospora parasitica* (Pers.) Tul.).—Like the last, this fungus attacks many plants of the cruciferous family, including most of the cultivated species. In India it has been found on turnips, cauliflower, sarson, toria, rai, radish, black mustard, and duan, and probably occurs on cabbages and other hosts as well.

The effect of the parasite on the host plant is somewhat similar to, though lesser than, that of *Cystopus candidus*. Owing to the very frequent co-existence of the two fungi (Fig. 119), it is not easy to separate their effects, but it may be said that *Peronospora* produces the greatest deformities in the stem, *Cystopus* in the flowers. The stem swellings may be limited or several inches long, in the former case often accompanied by abrupt bending of the stalk. The axis of the inflorescence is equally liable to deformity. The leaves and flowers are not often swollen, except the young ovary, which may be prolonged to a twisted body, two or three inches in length. More often the floral buds are atrophied, all the parts, sepals, petals, stamens, and pistil, being shrunk and almost colourless. If the attack is late, the fruits may be partly normal, partly deformed or atrophied, and a single fruit may be similarly affected in part only. There is never any trace of the violet colour produced so often by *Cystopus*.

The fungus is visible as a thin, greyish-white, downy growth, occurring in scattered patches on the under surfaces of the leaves in cabbage, cauliflower, and turnip (Fig. 20), and on the leaves, stem, and inflorescence in radishes. The upper surface of the leaf is marked by white spots corresponding to the downy growth below. In bad attacks, the spots may be so crowded that the leaf dries up, shrivels, and tears easily. In seedlings, the whole under surface may be evenly covered, and total infection of the young inflorescence is also found. Occasionally the roots are attacked. This has been observed in the radish and Swedish turnip in Europe. The tissues blacken and rot near the surface; oospores occur within the tissues and conidiophores form if exposed to the air.

The internal changes differ from those caused by *Cystopus* in many respects. The palisade cells of the leaf are not changed. In the stem, the cortex is less hypertrophied, the pith more. In the deeper layers of the cortex and in the endoderm and pericycle new layers may be formed by cell division. Only the cells just round the bundles become enlarged and thin-walled, the rest of the interfascicular sclerenchyma remaining unaltered. There is no interfascicular cambium. The cambium of the bundles remains active, the xylem vessels being somewhat enlarged and separated by radial bands of parenchyma, in which hyphae occur. The phloem is also increased. There are no accessory bundles. In general, the effect on the cells seems to be more destructive than in *Cystopus*, the chlorophyll being diminished and the cell contents more rapidly used up. There is not the same tendency to the formation of chlorophyll in unusual places as with the other fungus.

The mycelium of the parasite grows, like that of white rust, exclusively between the cells. The haustoria are large, elongated, club-shaped, often branched, and may nearly fill the nutrient cells into which they are chiefly sent.

After a period of vegetative growth, numerous erect, branched conidiophores emerge through the stomata of the under surface of the leaf and those of the stem and inflorescence. They may be absent in infected floral organs and then the tissues often contain oospores. The conidiophores arise directly from the mycelium and are flattened where they pass through the stomata, and also often twisted on themselves at the base. Several usually come through each opening. They are 200 to 300 $\mu$  long and bear conidia only on the branched tips, in strong contrast to the spore-beds of *Cystopus*. Towards the top, they bifurcate six or eight times, the final branches being long, slender, pointed, and terminating in a single conidium. Branching is at an acute angle, and the curved or undulating branches are often thickened a little above each fork.

The conidia are broadly oval, quite colourless, 24 to 27 by 15 to 20 $\mu$  in diameter, and fall very readily. They germinate in water by a lateral germ-tube, not by zoospores as in *Cystopus*, and infection occurs both by direct penetration of the epidermis and through the stomata.

Oospores are borne in the interior of the host tissues at a later period than the conidia, or sometimes (especially in hypertrophied parts) alone. They have also been found in the cavity of the ovary on hyphae emerging between the cells of the inner epidermis of the carpels. They resemble in their mode of formation those of *Cystopus* but differ in having a thick oogonal wall when mature. The persistent oogonium is irregularly rounded and swollen into crest-like folds, and is pale yellow. The oospore lies inside, almost filling the cavity, and is globose, yellowish-brown, and 30 to 40 $\mu$  in diameter. Germination in the genus *Peronospora* seems to be by a germ-tube in all the species observed, and not by zoospores as in *Cystopus*.

Infection may be either general or local. In the former case, all or most of the leaves may bear conidiophores and the inflorescence may be atrophied as already described, while some parts (especially the stem) may show no external change, though microscopic examination shows that the mycelium is in their tissues. Generalised infection seems to be only possible while the plant consists of young tissues, and this is why usually none but seedlings show totally infected leaves. Localised infection similarly occurs most easily in parts composed of young tissues, especially those still in active division. This appears to be the explanation of the frequent co-existence of *Peronospora* and *Cystopus*. In the swellings caused by the latter, the cells of the epidermis and cortex are dividing and thin-walled and readily give entrance to the *Peronospora*. In the same way, the young inflorescence may be wholly or partly infected, while the older stem and leaves below remain free.

It is as a leaf parasite that *Peronospora parasitica* causes most damage. Serious outbreaks have been recorded in cauliflowers in India, Europe, and (in plants under glass) in America. Occasionally it has been found to damage cabbages in the open; turnips and radishes are less often injured. In India, the cauliflower crop in the neighbourhood of Lahore was badly attacked in 1911-12. About 90 per cent. of the plants were infected and in bad cases two or three hundred conidial patches could be found on each leaf. The growers considered that the disease reduced the yield to less than half that of normal years. In combination with *Cystopus candidus*, the parasite has several times been reported in other countries to have injured this crop.

There is no information as to whether this fungus has specialised races on its different hosts. It has been noted, however, that it may be common on one host and absent from another in the vicinity, so that there is probably some degree of specialisation.

The treatment consists in clean weeding so as to remove all cruciferous weeds capable of harbouring the parasite, and in spraying with

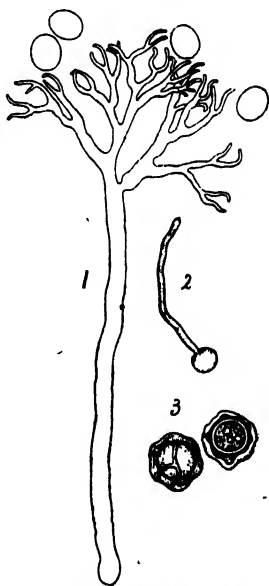


FIG. 121. *Peronospora parasitica*. 1, conidiophore and conidia from *Capsella Bursa-pastoris*,  $\times 285$ ; 2, germination of conidium from cabbage; 3, oospores. (2 after Duggar, 3 after Berlese).

Bordeaux mixture or potassium sulphide or some other fungicide. Spraying is only necessary in bad attacks. At the end of the season, all crop refuse should be carefully destroyed to get rid of the oospores; and rotation with non-cruciferous crops should be practised. An attack in young plants is reported to have been checked by a mulch of sawdust saturated with copper sulphate round the base of the plants.

**Powdery mildew (*Erysiphe Polygoni* DC.).**—The fungus which causes this disease is described above under peas (p. 253). In Great Britain, turnips (especially the Swede) are sometimes severely attacked by it, the oidial stage, long known as *Oidium Balsami*, being alone usually found. Perithecia were first observed in 1898 and proved to be identical with those of the pea mildew. A severe outbreak occurred in 1907 in the Chenab Canal Colony of the Punjab. Large areas were attacked about the New Year on irrigated land. The leaves were blighted and the roots dried up and became stringy. After a good fall of rain some recovery took place, new leaves being formed to replace the old.

The fungus grows on both surfaces of the leaves, producing conidia so plentifully as to whiten the boots and clothes of persons walking through the fields. The green colour of the leaf is lost; later, leaves and mycelium turn dirty brown. The conidia are borne on short, erect conidiophores as in the pea mildew, and are nearly cylindrical, with rounded ends, and 30 to 40 by 13 to 15  $\mu$  in diameter. The perithecial stage has not been observed on turnips in India.

Remedial measures do not appear to have been tried, but no doubt sulphur dusting would be as efficacious in this case as in that of the pea mildew.

**Blight (*Alternaria Brassicae* (Berk.) Sacc. = *Polydesmus exitiosus* Kuehn).**—This is one of the most widespread and destructive diseases of cruciferous crops in India, attacking turnips, cabbage, cauliflower, radish, sarson, toria, rai, *Brassica rugosa*, and dhan. In Europe, it appears to be most injurious to rape (*Brassica Napus* L.) but it also attacks turnips, radishes, and other Cruciferae. As stated above (p. 273) guar is sometimes affected in India by what seems to be the same disease. Varieties are also known on the carrot, melon, and other plants in some countries but have not been observed in India. Although first described in 1836 on English cabbages, the cabbage leaf-spot does not appear to be common in England; in parts of India, on the other hand, it is rare to find a mature plant without it.

In the neighbourhood of Pusa, sarson appears to suffer more from this disease than any of the other hosts. It was probably first recorded from Tirhoot in 1901, but the fungus which causes it was thought to be

new and described as *Sporodesmium Brassicae* Mass. It does not differ materially from the form found on turnips and other Cruciferae in Pusa and throughout northern India, and there is no need to give it a new name.

All the green parts of the plant are attacked, leaves, stem, and fruit. In sarson, toria, and rai, the pod is the seat of greatest damage in ordinary years (Fig. 122), but in years of severe outbreaks, the stem attack may be sufficiently intense to cause the whole plant to wither before many of the pods have matured. In other hosts, the injury is chiefly to the leaves (Fig. 21).

At Pusa, the disease appears, in sarson, usually in December and the attack reaches its height towards the end of January. At first, small, brown or blackish spots appear on the leaves. These multiply rapidly and later spread to the stems and pods. On

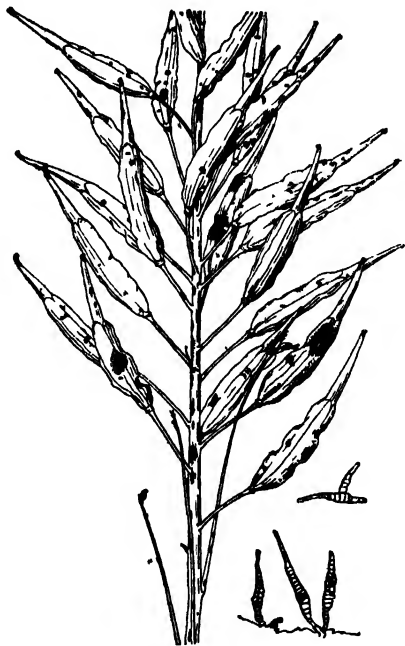


FIG. 122, Blight of sarson (*Alternaria Brassicae*) stem and pods with blight spots,  $\times \frac{1}{4}$ ; below, spores,  $\times 46$ .

the leaves and pods, they are punctiform at first, but gradually enlarge, remaining more or less circular in shape. On cabbage and its allies, the leaf spots are lighter in colour and may reach a diameter of about an inch, but on sarson they are generally smaller, and on the older leaves are mostly only from  $\frac{1}{4}$  to 2 mm. in diameter, and often slightly raised above the surface of the leaf. The spots on the stem of the latter host are at first linear and then expand

but remain usually elongated and with pointed ends. On young stems and pods they are often somewhat sunken, and, in severe attacks, the part between the spots and the tip of the stem or pod withers.

The fungus is found in the tissues underlying the spots and also in the cavity of the pods and surrounding the seeds. The internal hyphae run between the cells but can also penetrate them. The chlorophyll corpuscles become discoloured and the cell contents turbid and opaque. Later, the cell walls turn brown. After a limited growth within the tissues, further spread is usually checked and reproduction begins. The hyphae collect in stromatic masses beneath the epidermis; from the outer cells of these, branches pass through the epidermis, already partly disorganised from the effects of the stroma, to reach the outside and become sporophores. At first these stalks are hyaline, like the internal mycelium, and unseptate, then they turn brown below and develop cross walls. When old, the whole structure may be brown.

The spores are produced most freely on the stems and pods, being especially numerous, in sown, on the finer lateral branches. They may be in dense clusters or scattered on isolated sporophores. Typically, a single spore is borne on a rather short stalk, but several may develop in a chain: sometimes the chains are long (up to 10 spores) but in such cases each spore is usually dwarfed. In very damp weather, the stalk may give rise to a branched, hyaline hypha, bearing spores on the ends of the branches and on short lateral stalks. This is really a superficial spore-bearing mycelium.

The shape and size of the conidia vary considerably. They are usually somewhat bottle-shaped, but attenuated into a long beak at the tip and also slightly narrowed again at the base. They are divided by from 6 to 12 cross walls and also one or, rarely, two longitudinal walls at the broader part. The diameter is slightly narrowed at the cross walls. In colour, the mature spore is greenish-brown, the beak being nearly colourless. The size varies from 60 to 140 by 14 to 18 $\mu$ . Sometimes the beak is little developed, sometimes there are two beaks, and protuberances (sometimes even a second spore) may grow out laterally from some of the compartments. After falling, secondary spores may develop in the same manner. As the spores become detached very easily, it is rare to find chains of spores on spots examined after picking.

Germination is rapid, every cell being capable of giving out a germ-tube, though usually only some do so. Anastomoses may occur between branches of the germ-tube or between germ-tubes of different spores. Sometimes secondary conidia are borne on brownish branches from the hyphae given out by a primary spore. The spores retain their vitality for long periods, especially when kept dry. In Europe, successful infection has been obtained on turnips with spores gathered the previous year.

Infection occurs through the stomata, new spots appearing very rapidly and bearing spores in their turn three or four days after inoculation, if the weather conditions are favourable. Cultivators in Bihar state that severe attacks generally follow foggy weather. In Europe, moist, warm weather, or alternate periods of rain and sunshine, are said to favour the disease in rape-seed, especially if occurring when the pods are forming. It has been observed also in this crop that strong, well-grown plants are less readily injured than weakly ones, the pods, even when attacked, being able to ripen most of their seed.

The damage in the oil-seed crops is due to loss of seed and is much greater than in cabbages, turnips, and the like, where the leaf injury does



not usually reduce the value to any great extent. In bad attacks in sarson, the plants wither rapidly, especially the lateral twigs on which the pods are borne. This is due to the stem attack, which is sometimes sufficient to kill even the main stem. The crop is not uniformly affected, some plants withering much more rapidly than others; they can be seen even from a distance, scattered through the still green crop, about the middle of January. On closer examination, they are found to bear much smaller and, especially, thinner pods than normal plants, and the seed is often scanty and what there is remains unusually small even when fully ripe. The leaves fall prematurely, covered with spots, and the pods ripen before their time. In milder attacks, individual twigs alone may be killed and many of the pods escape serious injury.

Direct treatment by spraying does not appear to have been tried anywhere, and is not likely to be practicable on a large scale in India, as the cost would be high in relation to the value of the crop.

Besides securing vigorous plants by good cultivation, so as to assist the pods to develop even when an attack of the disease appears, it is probable that much loss may be avoided by early harvesting, as has been recommended in Europe when rape-seed is affected. It has been proved in the latter crop that the seed matures normally in the stack, if harvested at the moment when the seed of older pods is just beginning to turn brown, provided that the stacks are made so as to allow the pods to dry out slowly; at the same time the fungus ceases to grow after the plants are cut. The stacks are built with about 60 bundles, each 6 or 8 inches thick at the band, arranged conically in several layers, the lowest upright in the centre and sloping inwards at the outside of the stack, the upper layers still more sloped, so that their stalks protect the lower pods from the weather, and the top thatched with straw or stalks. The centre of the stack should be loose, to allow the pods to dry out, which they will do in fine weather in 12 to 14 days. Not only does the seed ripen in this way, but the oil content is said not to be diminished.

### **TOMATO** (*Lycopersicum esculentum* Mill.).

**Bacterial Wilt** (*Bacillus solanacearum* Smith).—Experiments in India and elsewhere show that this parasite is capable of attacking tomatoes, and though, so far, no cases have been observed in this country except those artificially produced, it is probable that some part of the wilt so well known to affect the crop in northern India will be found to

be due to this cause. It is discussed under potato (p. 274) and tobacco (p. 334).

**Blight** (*Phytophthora infestans* (Mont.) de B.).—As already mentioned (p. 286), the potato blight fungus also attacks tomatoes, the disease being similar on the two plants. The treatment is fully discussed under potatoes, spraying being the only effective measure with this crop.

**Fruit rots.**—As already mentioned under early blight of potato, the tomato fruit is attacked by a rot attributed to the fungus usually known as *Macrosporium Tomato* but apparently identical with *Alternaria Solani*. This disease has been several times observed in India, as has also (though more rarely) the leaf and stem disease caused by the same fungus. On tomato, the chief damage is to the fruit, but otherwise there is little to add to the description already given (p. 287).

A second fruit rot is caused by *Vermicularia Capsici*, a fungus which will be more fully described under chilli (p. 352). The widespread *Glomerella cingulata* has also been found to attack tomato fruits in the United States and elsewhere, while in India successful inoculations have been obtained by using *Glomerella piperata* from chilli, a species believed by some to be identical with the last. Other allied fungi found rotting the fruit in Europe and America are *Glomerella Lycopersici* Krueg. and *Colletotrichum phomodes* (Sacc.) Chester.

Early spraying with Bordeaux mixture would probably check most of these rots, but they have not hitherto been observed to cause sufficient damage to justify spraying. Damaged fruit should be picked and destroyed early.

**Wilt** (? *Fusarium Lycopersici* Sacc.).—Tomato wilt has not yet been specially studied in India, where it is common, and it is, therefore, not certain that it is identical with the disease caused by *Fusarium Lycopersici* in Europe, the United States, and Australia, the more so that at least two other species of *Fusarium* are said to attack the roots of the tomato.

The disease resembles the wilt of pigeon pea (p. 244) in all important respects, and the parasite found commonly present (but not proved to be its cause) is not easy to distinguish from *Fusarium udum*.

Where the crop is grown in the open, as in India, no treatment is possible except to avoid having tomatoes more often than once every three or four years on the same land. The fungus is a soil-dweller and can doubtless, like its allies, remain in land that has borne a diseased

crop, for a considerable time. Sterilisation of the soil, or its replacement from an uncontaminated source, has been employed with success in the large glass-house tomato industry in Europe and America.

**Root rot** (*Hypochnus Solani* Pril. & Del. and *Rhizoctonia* sp.).—*Hypochnus Solani* has been found attacking tomato plants in Kashmir, and the *Rhizoctonia* with small, black sclerotia is not uncommon on tomato in several parts of India. They have been fully described under cowpea (p. 262).

### BRINJAL (*Solanum Melongena* L.).

The only diseases of this crop so far observed in India are fruit rots, of which there seem to be several types, the *Rhizoctonia* with small, black sclerotia (p. 262), and a comparatively mild form of wilt which may possibly be due to *Bacillus solanacearum*, a parasite known to attack the crop in other countries (p. 338).

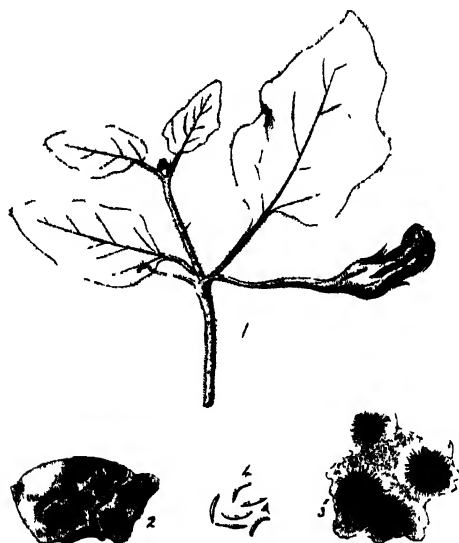


FIG. 123. Brinjal fruit rot (*Vermicularia Capsici*): 1, branch with rotted fruit,  $\times 4$ ; 2, part of rind of latter with sporodochia in clusters; 3, same more highly magnified; 4, spores,  $\times 200$ .

The commonest fruit rot in some parts of Bengal is that caused by *Vermicularia Capsici* (Fig. 123), a fungus more fully described under

chilli (p. 352). Others are caused by *Glomerella piperata* (p. 355), and a species of *Macrosporium* or *Alternaria* which seems to be that already described (p. 287) as *Alternaria Solani* (*Macrosporium Tomato* Cke. and *M. Lycopersici* Plowr.).

The most satisfactory method of checking these rots appears to be to pluck off and destroy the damaged fruit as soon as possible, so as to diminish spore formation.

### BHINDI (*Hibiscus esculentus* L.).

**Mildew** (*Erysiphe Cichoracearum* DC.).—The mildew of this crop has been found on several occasions in India. It is known also in Egypt and south-eastern Europe on the same host, and in many other parts of the world on other plants. The form on cucurbits in India is figured below (p. 314, Fig. 128).

The fungus forms a floury coating on the leaves, chiefly on the upper surface, covering all or a considerable part of the leaf. Affected leaves dry up and lose their green colour, just as in the powdery mildew of peas. The effect of a severe attack is to reduce both the size and number of the fruit.

The mycelium is superficial except for the haustoria, which penetrate the epidermal cells as in the mildews of wheat and peas. From the recumbent hyphae, short conidiophores arise more or less at right angles to the surface, and bear chains of spores of the usual oidial type. The spores are barrel-shaped and measure 24 to 30 by 15 to 20 $\mu$ .

Perithecia seem to have been observed only on specimens collected in the Balkans, where the crop is extensively grown. They occur on the under side as roundish, dark bodies, about 80 to 140 $\mu$  in diameter, and with numerous densely-interwoven, septate, brown appendages, which are twice to four times as long as the diameter of the perithecium. The asci are numerous, ovate, 58 to 90 by 30 to 50 $\mu$  in diameter, and contain each 2 spores, which are usually about 24 by 14 $\mu$  in diameter, hyaline, oval, and continuous.

The treatment is the same as that of the pea mildew (p. 254).

### KACHU (*Colocasia antiquorum* Schott.).

**Blight** (*Phytophthora Colocasiae* Rac.).—Only one serious disease of this common Indian garden crop has as yet been reported in India. It is known also in Java, the Philippines, and Formosa, and probably throughout south-eastern Asia. In India, it occurs sometimes on the wild varieties of *Colocasia* which grow in moist situations, and pure cultures of the fungus have been found capable of attacking seedlings of *Gilia* (an exotic garden flower) and wounded leaves of potato and perhaps also tomato, but no other hosts are known.

The disease first shows itself ordinarily on the leaf, the earliest attacks occurring in August or September at Pusa, in the form of small, dark, roundish specks, which widen rather rapidly by centrifugal growth. The spots may remain circular or become oval or irregular. Several often coalesce, and in bad attacks a very large part of the leaf area may become involved. In the early stages, drops of a clear, yellow liquid ooze out from the surface of the spots; later, the central portion assumes a yellowish-brown colour, dries up, and may become perforated. The margins are often beautifully zoned, in different shades of brown, green, and yellow; towards the periphery these zones reveal, on careful examination, a delicate white haze which is caused by the sporangial stage of the fungus. This external development of the parasite is, however, much less prominent than in the allied potato blight, and is sometimes difficult to detect with the naked eye. As the spot widens, sporangia continue to be produced at progressively increasing distances from the centre.



FIG. 124. Colocasia blight (*Phytophthora Colocasiae*): young inflorescence and leaf severely attacked,  $\times 8$ .

The above may, in mild cases, constitute the whole extent of the attack. Frequently, however, the petioles become infected, with more serious results. The tissues at the infected point often become so softened as to be unable to bear the weight of the large leaves, which fall over and may even break off altogether. Extension may continue down to the sheathing base of the petiole and probably into the short stem, though this has not been actually observed. The inflorescence and corm are

also attacked, the former in the same way as the leaves, the latter possibly by extension down inside the tissues but more probably from spores shed on the soil. In a severe attack, all the leaves may be lost and the plant killed. In less severe cases, the young leaves may be sound or only slightly involved, while the older ones are covered with patches of varying extent or are hanging rotted on their stalks. The corms may be entirely lacking or, if formed, may be small and shrunken. Where only isolated spots occur, there is little apparent injury to the plant and the corm matures normally. The intensity of the attack depends, as in potato blight, on the atmospheric conditions prevalent during the growth period of the host, being much greater in years of abnormal humidity; it is also greater in shady, sheltered places than in open fields.

The corm rot is sometimes prevalent before the crop is lifted, but more usually develops subsequently in storage. It resembles that of potato tubers caused by *Phytophthora infestans* and, so far, has only been found to cause considerable damage in unusually wet years.

The parasitic mycelium grows within the tissues, the hyphae being large, unseptate, and copiously branched. In the leaf they grow between the cells, except in the epidermis, where they sometimes cross a cell when entering or leaving the leaf. Longish, slender, usually unbranched haustoria are sent into the cells in considerable numbers (Fig. 16). Neither in the leaf nor in the petiole have hyphae been found to enter the fibro-vascular bundles. In the corm, on the other hand, not alone are hyphae found in the storage cells, the mycelium being rather within than between the cells, but the bundles are commonly penetrated. The effect on the cells is to cause the gradual disappearance of the green colouring matter and the collection of the cell contents into a shapeless, grumous mass. Later on, the starch is destroyed and the cells turn brown.

From the internal hyphae, branches grow out to the surface to form sporangia. These are found at the margins of the spots on both surfaces of the leaf and on the petiole and inflorescence. They come out in successive crops as the spots widen. Similar sporangia can be induced to form on the corm in water or moist air, for some months after growth elsewhere has ceased, and no doubt the storage rot is spread in this manner, as in the potato blight.

The sporangia are borne on very short stalks, not often more than  $50\mu$  in length, and exceedingly narrow (sometimes as little as  $1.5\mu$  in diameter) at the tip. The stalks emerge singly or in small clusters not only through the stomata (the usual mode of emergence in the Peronosporaceae) but also through or between the epidermal cells (Fig. 125). They are usually unbranched. The sporangia are colourless, elliptical or pear-shaped, sometimes curved, and very irregular in size, measuring from 38 to 60 by 18 to  $26\mu$  in diameter. They are borne singly at the tip of the stalk and break off, when ripe, with a short piece of the stalk attached, like a tail. The wall is thin and smooth, and at the free end is a broad, blunt papilla, which swells up and softens after a short time in water and then opens to set free as many as 20 zoospores. These swim freely for a time, by means of two cilia, and then settle down, become covered by a wall, and germinate by a germ-tube, the whole process often taking not more than an hour from the liberation of the zoospores. As in most of its allies, temperature influences zoospore-formation to a marked degree. At temperatures above  $90^{\circ}\text{F}$ . it is checked, while below  $80^{\circ}$  it occurs

freely; the lower limit is not known. In many cases, especially if the sporangia do not come in contact with fresh water as soon as they ripen, they fail to form zoospores but germinate directly by a hypha, which usually grows from the papillate end of the sporangium. This hypha may form a small, branched mycelium or may bear secondary sporangia after a short growth. Both the germ-tubes from the zoospores, and the hyphae which grow from those sporangia which have failed to form zoospores, are capable of infecting the *Colocasia* plant, and it can be readily understood that the powers of dissemination of the parasite in moist weather are very considerable.

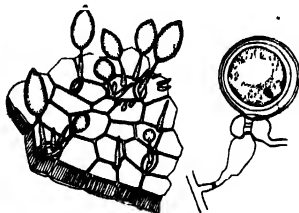


FIG. 125. *Phytophthora Colocasia*: surface of leaf with sporangia,  $\times 160$ ; oospore showing antheridium surrounding the stalk,  $\times 465$ .

No other spore form has been found in the fungus in its normal home on the *Colocasia* plant. It has, however, been cultivated artificially on specially prepared nutrient substances, and under these conditions two other types of spore may develop. One is a thick-walled, spherical body, varying from but little more than the breadth of the hypha to over  $30\mu$  in diameter, and formed anywhere on the mycelium by the simple swelling up of part of a hypha, which is then cut off by one or two septa, according as it is terminal or in the middle of a branch. It is not a true spore, but belongs to the vegetative type of propagating organ known as a chlamydospore. It is doubtless capable of preserving its vitality for long periods and of giving rise to new hyphae after some months of suspended activity, as has been proved to occur in other cases, though this has not been actually observed in the *Colocasia* fungus.

The other spore form which has been developed in culture is a sexually-formed oospore (Fig. 125), closely resembling in its characters and mode of formation the oospore of *Phytophthora infestans* (p. 281). The oogonia are nearly round, of a yellow colour, and measure from  $24$  to  $35\mu$  in diameter. Attached to the base is an antheridium, which persists for a long time after the oospore has been formed. The latter is spherical and measures from  $20$  to  $28\mu$  only, so that it lies loose in the oogonium. Though they have not been germinated, it is certain that the oospores form a resting stage of the parasite and are capable of germinating long after the sporangial stage has ceased. If they and the chlamydospores occur in nature, they must serve the purpose of enabling the fungus to persist during that part of the year when the host plant is not available. This probably takes place in the soil. Another possible way in which the disease is carried over is by the mycelium in the corms which, as has already been mentioned, is capable of producing sporangia long after the leaf stage of the fungus has ceased.

Infection takes place with great rapidity when active zoospores are placed in a drop of water on the leaf. The germ-tubes pierce the epidermis at any point and pass down to the internal tissues (Fig. 17), causing, in some cases, visible spots within six hours. New sporangia may arise from these spots in three days. In the same way, the hyphae from sporangia which germinate directly can penetrate the leaf, and it, as well as the wounded surface of the corm, can be penetrated by the ordinary aerial growth of hyphae obtained by placing a diseased leaf in a saturated atmosphere. The fungus is a virulent parasite, provided the surrounding conditions are favourable to its growth.

The milder form of the disease is found in most parts of India but is especially common in the eastern provinces. Severe attacks have only been observed at Pusa in exceptionally wet years.

Treatment by spraying is only likely to repay cost when the attack is severe. Bordeaux mixture is reported to have given good results in Formosa. It should be applied as in potato blight. In the early stages of attack, spotted leaves should be removed and destroyed. Corms kept for seed should be gone through at intervals, and any that show signs of rotting removed. At the time of planting, only sound corms should be selected. This is probably the most important point in the control of the disease, as provided that a sufficient rotation is practised, the fungus is likely to disappear from the soil unless reintroduced in infected corms. Planting in shady localities should be avoided.

### CASSAVA OR TAPIOCA (*Manihot utilisima* Pohl.).

**Leaf spot** (*Septoglœum Manihotis* Zimm.).—This crop is extensively grown in the south-western parts of India, especially in Travancore. The leaf spot is common, but as it occurs chiefly on the older leaves does not seem to do much damage. It is known also in Java, Ceylon, and East Africa.



FIG. 126. Leaf spot of cassava (*Septoglœum Manihotis*): part of leaf with spots,  $\times 4$ ; conidiophores and conidia,  $\times 200$ .

The leaves show dark green spots which soon turn brown, extending from the margin towards the main veins. They are up to  $\frac{1}{2}$  inch in diameter and are dotted with greenish-black pustules on both surfaces, representing the fructifying stage of the parasite.

The fructifications consist of stromatic collections of mycelium under the epidermis, with a dense layer of conidiophores (basidia) on the outer side which rupture the epidermis and project a little way above the surface of the leaf. The stomata are 40 to 100  $\mu$  in diameter, black, and when mature rather cushion-shaped.

The sporophores are short (not usually exceeding 50  $\mu$ ), unbranched, and septate only at the base. They each bear a single conidium on the tip. The conidia are cylindrical, straight or irregularly curved, hyaline, with 0 to 9 (usually 2 to 5) septa, and measure from 20 to 90 by 4 to 8  $\mu$ . The upper end is rounded, the lower somewhat narrowed

and marked by a flattened plate showing the point of attachment to the stalk. Their germination has not been observed.

No case has as yet been reported in which treatment was required, but an allied (perhaps identical) leaf disease of this crop in the West Indies is said to be readily controlled by spraying.



**THE CUCURBITS** (Gourds, Melon, Cucumber, etc.).

**Downy mildew** (*Pseudoperonospora cubensis* (B. & C.) Rost.).—This parasite is an interesting example of a fungus, described first on what seems to have been a wild plant, in an isolated part of the world, and considered of no economic importance, which by its gradual spread to other countries and the damage that it has caused on new hosts has won for itself a position amongst the major enemies of cultivated plants.

It was first described from Cuba in 1868. Nothing more was heard of it until 1889, when a new blight on greenhouse cucumbers in New Jersey (United States) was found to be due to this fungus. It subsequently transpired that it had been collected the previous year in Japan. In a few years it had spread over most of the eastern and southern States, and it was collected in Ecuador (South America) about 1891. It was not, however, until 1899 that it was reported in Europe; in that year it was stated that it had recently been observed in England. It was collected in Brazil in 1900, Russia and Java in 1902, Austria, Italy, and East Africa in 1903, China in 1908, and Australia and India in 1910. When it first reached India is not known, but it was certainly not in Pusa much before 1910, as it has appeared regularly every year since and could not have escaped notice for long. An unidentified *Peronospora* was recorded on cucumber in Ceylon in 1901, and as the present is the only member of the family known on this host, it is quite possible that the disease has reached us from the south.

In one or other of these countries, it has been found on almost every cultivated cucurbit, perhaps most frequently on cucumbers. At Pusa it occurs on species of *Luffa* and *Trichosanthes*, in the Punjab on melons, and it is also known in Bombay.

The fungus attacks the leaves, causing at first pale yellow, angular patches, which deepen to a brownish-yellow as they grow older. The shape is very characteristic, the limitation of growth by the veins of the leaf causing the spots to assume a sharply defined figure. In late stages, much of the leaf may become covered, the spots uniting into larger patches. As these dry up, the whole leaf turns yellow and decays. The rot is not usually a wet one as in potato blight; the leaves simply wither and fall. As the young parts do not at first show the disease, the leaves are lost from the base upwards, and a tuft towards the tip of the creeping stem may be all that is left on the plant. On the under surface of the spots, careful examination may reveal the fine downy outgrowth of

sporangiophores, which is often scanty and hard to see. The fruit is not directly attacked, but once the leaves are well infected, few fruits are developed and those that appear are often small and misshapen.

The mycelium consists of hyaline, branched, irregular non-septate hyphae, running between the cells of the leaf mesophyll, which are penetrated only by short ovate haustoria. From these hyphae the sporangiophores arise chiefly on the under surface, through the stomata but sometimes boring directly through the epidermal cells. Usually one or two

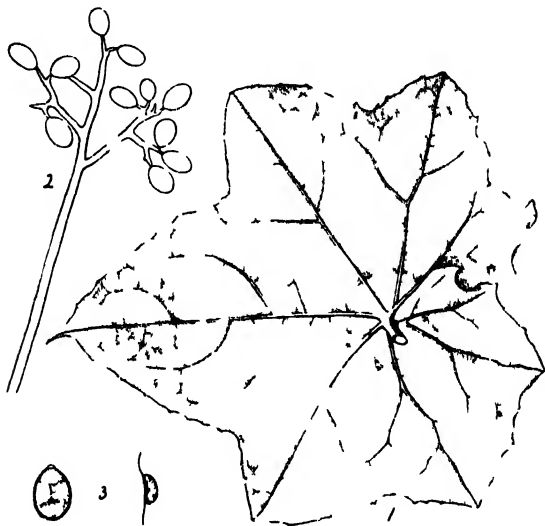


FIG 127 Downy mildew of cucurbits (*Pseudoperonospora cubensis*) 1 affected leaf of Luffa,  $\times \frac{1}{2}$  2 sporophore and sporangia from Trichos anther,  $\times 260$ , 3 sporangium and zoospore (3, after Clinton)

sporangiophores come through each stoma but there may be as many as five or six together. They are mostly 200 to 300  $\mu$  long, varying, however, considerably; the breadth is 5 to 9  $\mu$  below the first branches. The upper third is 3 to 5 times branched in a roughly dichotomous manner, the branches being at an acute angle and one generally tending to continue the direction of the main stem. They are slightly curved. The ultimate branches taper to a rather blunt apex, on which is borne a single sporangium.

The sporangia are hyaline when immature, then smoky grey or purplish and in mass greyish black. They are ovate or ellipsoidal, with a small hyaline papilla at the free end and when ripe and average 23 to 30 by 16 to 20  $\mu$  in diameter. They fall off when mature and germinate in water, usually by giving out zoospores but sometimes by a germ tube. The zoospores emerge singly as a rule and are of the usual Peronosporaceous type, about 10 or 12  $\mu$  in diameter after coming to rest. Infection occurs from either surface of the leaf, the germ tube being capable of penetrating through the epidermal cells as well as  $\sigma$ .

entering through the stomata. Growth does not seem to be rapid, the new conidiophores sometimes taking over a week to appear.

Oospores are not known, and as the plants are annuals and the sporangia short-lived, it is somewhat of a mystery how the parasite persists from season to season, especially in parts of the world where there is only one season for growing the host plants. In Pusa there are suitable hosts during most of the year and there may be an almost continuous succession of living sporangia. Immature oospores are said to have been found in Russia on very old rotting leaves, but there is some doubt that they really belonged to this fungus.

There is no evidence that the races on the different hosts are specialised, such observations as have been made indicating rather that spores from one host will readily infect another.

The spread of the disease is greatly influenced by the humidity and perhaps also by the temperature. In dry weather, few conidiophores are formed and the spores rapidly lose their power to germinate. Low temperature probably favours germination. Cloudy or foggy, cool days, with heavy morning dews and little sun, are likely to be the most dangerous. At Pusa, rainy season cucurbits are chiefly attacked. In Florida (United States) it is said that the fungus grows all the year round on various hosts, and some hold that the annual attack in the more northern States is due to spread each year afresh from the south. If such extensive spread really can occur, the great diversity of climate and season in different parts of India and the frequency of suitable hosts would probably ensure the continuous growth of the parasite.

Cucumber and melon plants are readily killed in other countries. The hardier species infected at Pusa have not been seriously damaged up to date. As the disease is so recent there is no information of the amount of damage it causes in other parts of India. Elsewhere it is perhaps the worst fungus disease of cucurbits known; melon and cucumber fields may be destroyed in a few days; and losses of two-thirds of the late varieties of cucumbers have been recorded in parts of the United States and of 80 per cent. of melons in Austria.

Spraying has been effective in checking the attack on cucumbers in America, where two or three other diseases often co-exist with the mildew and afford additional arguments for this treatment. On melons, the results have not been satisfactory on the whole. The different hosts vary greatly in susceptibility, cucumbers being more readily damaged than melons, and melons than vegetable marrows. The hosts attacked in Pusa have been little injured so far, but spraying might be necessary if the more susceptible species largely grown in certain districts become infected. Bordeaux mixture has been found rather hurtful to some

cucurbits at the ordinary strength and may preferably be made up with 2 lb. copper sulphate, 2 lb. lime, and 50 gallons water. Spraying should commence about the time the disease first shows, and be continued at intervals of 14 days or so.

**Powdery mildew** (*Erysiphe Cichoracearum* DC. and *Sphærotheca Humuli* (DC.) Burr. var. *fulginea* Schlecht.).—Two species of Erysiphaceæ attack the cultivated cucurbits in India, *Erysiphe Cichoracearum*, which has been already described under bhindi, and *Sphærotheca Humuli* var. *fulginea*, a fungus which is parasitic on a large number of plants (mostly weeds) in many parts of the world.

*Erysiphe Cichoracearum* on cucurbits agrees with the description on p. 306 above, except that the appendages of the perithecium are



FIG. 128. Powdery mildew of *Oephalandra* (*Coccinia*) *indica* (*Erysiphe Cichoracearum*): 1, leaf with white mycelial patches,  $\times \frac{1}{2}$ ; 2, conidiophore and conidia,  $\times 120$ ; 3, a perithecium with appendages,  $\times 130$ ; 4, asci with ascospores,  $\times 130$ .

often short. It has been found in the perithecial stage on *Cephalandra* (*Coccinia*) *indica* at Cawnpore, and on the same host as well as *Trichosanthes dioica* and *Momordica Balsamina* at Pusa. Many of the oidial forms of powdery mildew so common on most of the cultivated cucurbits of the plains are doubtless also to be referred to this species.

*Sphærotheca Humuli* var. *fulginea* has so far only been found bearing perithecia on *Lagenaria vulgaris* in the United Provinces, and *Cucurbita moschata* in Kashmir. Some of the oidial forms on other cucurbits, however, probably belong to this species. The disease resembles that caused by *Erysiphe* so closely that no general distinguishing feature has been observed.

The mycelium occurs on both surfaces of the leaf, but chiefly above, forming more or less persistent, large or small patches, which are dusty white when bearing conidia.

The perithecia occur as scattered or crowded, minute, black points on, or partly immersed in the mycelium. They measure from 40 to about 100 $\mu$  in diameter and have short, pale brown, bent, and interwoven appendages. Each contains a single ascus, roundish or broadly elliptical in shape, and with 8 ascospores. The latter measure 20 to 25 by 12 to 15 $\mu$ , and are hyaline, oval, and continuous.

The treatment of these mildews is the same as described for pea mildew (p. 254).

**Root rot** (*Hypochnus Solani* Pril. and Del. and *Rhizoctonia* sp.).—The form with small, black sclerotia (see p. 262) occurs on melon, Cucurbita, and Citrullus, while the *Hypochnus* has only been found on *Trichosanthes*. They are described above, under cowpea.

### CELERY (*Apium graveolens* L.).

**Leaf spot** (*Cercospora Apii* Fr.).—This disease has been found occasionally in India and is common in Europe and America, where it also attacks carrots and parsnips. It is called "early blight" in the United States.

The affected leaves become covered with dark yellow spots, up to  $\frac{1}{4}$  inch in diameter and with a slightly raised rim. In the centre, the conidiophore clusters appear as brown, powdery dots. In bad attacks the spots are large and indefinite, and, as the leaves die, they may become almost completely covered with spores. They are first visible on the lower leaves but extend rapidly over the plant, especially in warm, moist weather. The actual injury inflicted appears, however, to be greatest in hot, dry weather, as then the plants are less able to resist the attack.

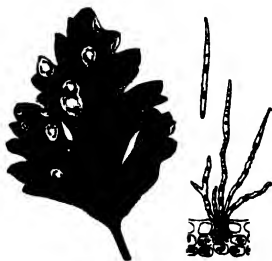


FIG. 129. Celery leaf spot (*Cercospora Apii*): leaf with spots, ; conidiophores and conidia (after Delacroix).

The fungus mycelium occurs within the leaves, passing between the cells, which are killed and turn brown. Tufts of conidiophores emerge through the stomata all over the spot. They vary considerably in length according to the weather, being usually about 40 to 60 by 4 to 5 $\mu$ , but sometimes up to 150 $\mu$  long. They are brown, undulate, with one or two septa near the base, and generally unbranched.

The conidia, which are borne singly on the ends of the conidiophores, are very pale green, rounded at the base, attenuated above, with up to 12 septa, and usually 50 to 80 by 4 to 5 $\mu$  but in warm, moist weather much longer, up to even 200 $\mu$  in some cases. They maintain their vitality for many months, and germinate by one or more germ-tubes which can readily penetrate the leaf and start a new spot.

The treatment by early spraying with Bordeaux mixture has given good results in the United States, where sulphuring has not been successful on the whole. Ammoniacal copper carbonate has also proved satisfactory. Trenching, and the resulting better aeration of the surface soil and control of the water-supply, is of great importance. Shading has been also recommended, as shaded plants are said to resist the attack better. Removing infected leaves might delay and diminish the progress of the disease.

### CHUA (*Amaranthus paniculatus* L.).

**White rust** (*Cystopus Bliti* (Biv.) Lév.).—The amaranths are cultivated somewhat extensively in India, some as food grains, others as green vegetables or pot-herbs. The present species is one of the most important food grains of the hill peoples, from the Himalaya to South India and Burma; it is also found as a cold-weather crop in the plains.

It is sometimes attacked by a disease allied to the white rust of the Brassicas (p. 291). The leaves are chiefly affected and bear numerous small, white blisters on the under surface. These are the sporiferous sori of the parasite, which form under the epidermis, raising it up and ultimately rupturing it. The corresponding upper surface is marked by pallid spots. No deformity is caused so far as has been observed, but attacks during the flowering period have not been seen and if, as is probable, the inflorescence is also involved, the effects may be more considerable than on the leaves.

The fungus agrees in most points that have been made out with *Cystopus candidus*, except in the spore characters. The sporangia of *C. Bliti* are marked by an annular, thickened, horizontal band on the inside of the wall, about half way up the spore. The end spore of the chain is smaller than the rest, round, with a thick wall and yellowish contents and is incapable of germination. The others are colourless and round-oblong or egg-shaped, but with the ends (especially the base) somewhat flattened. They are narrower below than above and measure 15 to 20 by 14 to 18 $\mu$ .

Oospores are found in the leaves, as the spots begin to wither. They are dark brown, spherical, and with a thick epispore clearly marked by a fairly regular, 5 to 6-sided reticulation, with meshes 5 to 6 $\mu$  across. They measure 55 to 65 $\mu$  in diameter.

The fungus is said to permeate the whole plant, but little is known of its life-history or mode of infection, except by analogy with the other members of the genus.

In the absence of more accurate information, suggestions for treatment cannot be given, beyond attempting to destroy the oospores by burning the infected debris. Probably the crop is not so seriously damaged as to allow of much added expense in employing remedial measures.

Several of the other cultivated amaranths are attacked by the same fungus in India, the most interesting of which is *A. Blitum*, a pot-herb. In this host, the oospores are found in the stem, not in the leaves, and differ from those described above in being of lighter colour and with larger, less regular meshes on the wall. For this reason the parasite has been regarded by some as a distinct species. In Italy, *A. silvestris* similarly bears the oospores in the branches, where gall-like swellings are produced, two or three times the size of the normal stem, of a bright red colour, and variously bent or flexed; while on *A. retroflexus*, oospores occur in the leaves or more commonly in the inflorescence, causing the parts to turn red without hypertrophy and to stand out in strong contrast to the rest of the plant.

### BATHUA (*Chenopodium album* L.).

**Downy mildew** (*Peronospora effusa* (Grev.) Ces.).—This plant is a common weed in many parts of northern India, but is also grown as a green vegetable, like spinach. It is very liable to the mildew, which, however, does little apparent damage.

The fungus occurs as a fine, woolly, grey-violet growth, composed of great numbers of conidiophores, on the under (rarely upper) surface of the leaves. The latter are marked by bleached or yellowish patches, sometimes covering one-third to almost the whole of the leaf, slightly raised on the upper side and hollowed below. The affected patches wither early and the leaves are checked in their growth and sometimes killed. Severe attacks leading to death of the plant have not been observed, but the loss of leaf may cause stunting.

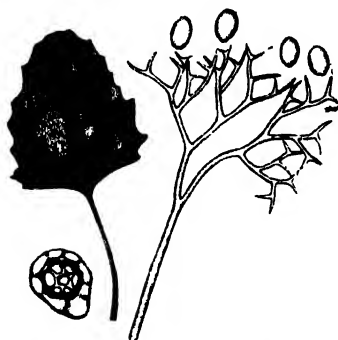


FIG. 130. Downy mildew of *Chenopodium album* (*Peronospora effusa*): leaf with grey patches,  $\times \frac{1}{2}$ ; conidiophore and conidia,  $\times 200$ ; oospore (after Berlese).

Within the leaf, the mycelium ramifies between the cells, sending branched, finger-like haustoria into them. Conidiophores emerge through the lower stomata in groups of 4 or 5. They are 200 to 400 $\mu$  long by 8 or 9 $\mu$  broad and branched at the top repeatedly (3 to 6 times) in a dichotomous manner. The branches are usually straight or slightly

flexuous, the end branches being divergent at nearly a right angle, more or less equal to one another in length, and never convergent at the tip or pincer-shaped. At the tip of each branch is an oval conidium, measuring 22 to 33 by 15 to 21  $\mu$ , with colourless contents, and a very faintly tinged violet wall. There is no papilla at the free end, but after falling a short knob may be noticed at the base. Germination occurs by a germ-tube, usually from the side of the spore, which can enter directly into the epidermal cells if sown on a suitable host and, after crossing them, reaches the intercellular spaces below, where it continues its growth to form a purely intercellular mycelium.

At a later period, oospores are produced within the leaves. The oogonia are thin-walled, colourless, and do not persist long after the oospores form. The latter are round, 30 to 36  $\mu$  in diameter, and with a thick, brown episore, which is more or less irregularly reticulate or crested. Germination does not seem to have been observed, but is presumably by a germ-tube as in allied species.

The attack is not usually epidemic, only single plants here and there being strongly affected. As the conidia are short-lived, there is no reasonable doubt that the parasite lives over from one season to the next by means of its oospores.

A closely allied species (*P. farinosa* (Fr.) Keiss.) attacks *Chenopodium album* and related plants in Europe, Asia, and America, but has not yet been seen in India. *P. effusa* is mostly found on spinach outside India but has been found on *Chenopodium album* in America. It has been stated that it can be controlled by spraying. Sulphuring is also effective, and easier to apply in small plots. In India, the crop is of too little value to justify spraying and treatment is probably not practicable. All the old debris of infected plants should be burned, in order to destroy the oospores.

### KULFA (*Portulaca oleracea* L.).

**White rust** (*Cystopus Portulacæ* (DC.) Lév.).—A disease allied to those already described on the Brassicas (p. 291) and amaranths (p. 316) is often found on this plant, which, like the last, is used as a green vegetable in parts of India. No other plant but *Portulaca* is known to be attacked by the fungus, but on this host it occurs in Europe, America and Africa.

The parasite is visible chiefly on the leaves, less often on the stem and inflorescence. It causes raised, white or pale yellow blisters, of a roundish or irregular outline, on the upper surface. These result from the development of spore-beds under the epidermis. The latter is ultimately ruptured and a whitish powder, composed of sporangia, liberated. Little deformity is caused and there is no obvious hypertrophy of the affected parts.

The mycelium is intercellular, richly provided with vesicular haustoria, and found in almost all parenchymatous parts of the plant, except the roots. Its structure (and



that of the sori) is just as in *C. candidus* (p. 291). The sporangia are in chains, the end roundish-oblong, with a thick, yellowish wall, and larger ( $20-22\mu$ ) than the others, which are elliptical, broader above than below, with a colourless, moderately thick, even wall, and 14 to 17 by 12 to  $14\mu$  in diameter. The end spore is sterile, the others germinate in water like *C. candidus*, but with rather larger zoospores.

Oospores are found in quantity in the neighbourhood of the sori. They are round,  $45$  to  $60\mu$  in diameter, with a thick, bright brown episore, beautifully marked by a regular pentagonal or hexagonal reticulation, the meshes of which are up to  $7\mu$  across and have a little crest or papilla in the centre of each. Their germination resembles that of *C. candidus*.

Infection occurs, so far as is known, in the same manner as in the white rust of the crucifers.

The remarks regarding treatment under the last head apply equally here.

### GROUNDNUT (*Arachis hypogæa* L.).

**Tikka** (*Cercospora personata* (B. & C.) Ellis, = *Septoglæum Arachidis* Rac.).—This is one of the major diseases of cultivated plants in India, being responsible at times for very great destruction of the crop. It is known also in the United States, West Indies, Surinam, Paraguay, the Congo, East Africa, Ceylon, Java, Malaya, China, the Philippines, and Australia.

The disease usually appears when the crop is between one and two months old. The lower leaves are the first to be attacked; dark spots, surrounded by a bright yellow ring, come out in large numbers on the green leaves; a few also occur on the petioles and stem. The number of spots on a single leaf may be from one to a dozen or more, and their size from one-tenth to one-third of an inch in diameter, the shape being generally round. Sometimes two or more run together, and any part of the leaf, including the veins, may be involved. The remainder of the leaf slowly loses its green colour, but before this is gone it falls to the ground. The shedding of the leaves is the most striking feature of the disease; it is apparently due to some poisoning action of the parasite, as even those with only a single spot often fall prematurely. After the disease has been in progress for a week or two, affected plants can be at once recognised by the litter of fallen leaves round their base.

In the later stages, the effect is not unlike that of potato blight. The field which, earlier in the season, had been densely covered by the

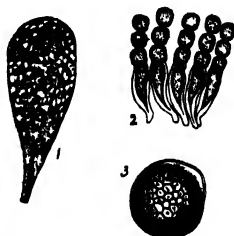


FIG. 131. White rust of *Portulaca* (*Cystopus Portulacæ*): 1, leaf with sori, nat. size; 2, part of a sporangial bed,  $\times 200$ ; 3, an oospore,  $\times 200$ .

green foliage, shows a mass of fallen, withered leaves, from which the bare stalks, with perhaps still a few young leaves at the tip, stand out.

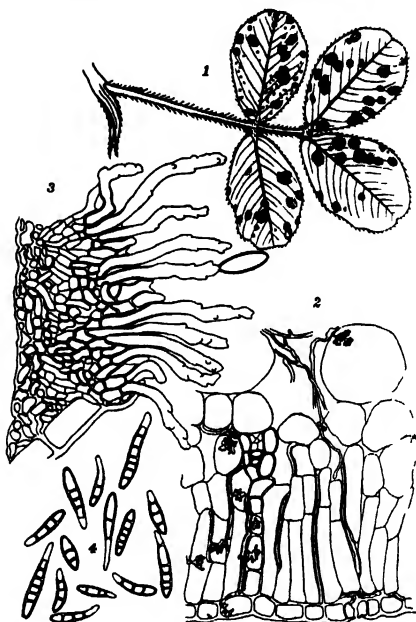


FIG. 132. Tikka disease of groundnut (*Cercospora personata*): 1, leaf with spots; 2, section of same showing hyphae and haustoria,  $\times 240$ ; 3, stroma and conidiophores,  $\times 475$ ; 4, conidia,  $\times 240$ .

Naturally, when the attack begins early, the plant is unable to mature its nuts. Those that have begun to form as the attack reaches its height, cease development, and at harvest are found shrivelled and loose in the shell. If they have reached a certain stage of maturity before the disease becomes severe, the reduction of the total yield of nuts is less marked, as the loss of leaf occurs too late to check their development. In many cases, however, losses from one-third to one-half have occurred in infected fields.

The mycelium of the parasite is found in the plant tissues in the neighbourhood of the spots. It is composed of slender, branched hyphae, which run chiefly between the leaf cells and send characteristic, lobed haustoria into them (Fig. 132, 2). Cells so invaded wither and collapse. After growth has thus continued within the leaf for a week or two, the hyphae collect in masses under the epidermis of both surfaces but chiefly underneath, and form small, cellular cushions of a brown colour. The superficial cells grow out into

cylindrical protrusions, which by their pressure rupture the epidermis and appear on the surface as conidiophores (Fig. 132, 3). The cushions (stromata) are usually grouped in concentric circles, which have a powdery appearance from the loose mass of spores with which they are covered.\*

The conidiophores are thick (up to about  $8\mu$  in diameter), short, unbranched, and unseptate, and are marked by pronounced angular bends, each of which represents the point of attachment of a spore. The spores are formed singly at the end of the conidiophore but the latter continues to grow on, leaving the spore attached to a bend at the side of the hypha. Each spore falls off as soon as ripe.

The spores are irregularly cylindrical, straight or curved, rounded at the free end and slightly flattened at the base, ashy-grey to light brown in colour, and divided by cross septa into from 3 to 8 cells (usually 4 or 5). They measure from 20 to 55 by 6 to  $8\mu$ . They germinate readily in water or very damp air, putting out usually a single germ-tube from near one end of the spore. Dried spores have been found to germinate after six months and there is evidence that spores can remain alive in the soil long enough to infect the succeeding crop. Inoculation experiments show that infection can be easily produced by sowing the spores in water on the leaf, new spots appearing in from four days to about a week.

The disease is spread chiefly by the wind, which blows the spores from leaf to leaf. Insects have also been proved to convey the spores on their bodies and in their alimentary canals. New attacks seem to come from the soil in which the spores have remained from a previous crop. No other stage than the conidial is known, and no other plant than groundnut has been found to harbour the fungus near Poona, where a very careful search was made owing to the recurrence of the disease for several successive years. In the United States, the fungus is said to pass the winter in the conidial stage on fallen diseased leaves; new crops of conidia can also be formed on old conidiophores on such leaves. In India, the rapid decomposition of organic matter causes plant debris to disintegrate too quickly for this method to be possible. Another method by which the disease is said to be carried over is in the seed, but attempts to check it by seed disinfection failed at Poona, when the crop was grown near plots diseased the previous year. Still seed from a diseased crop, treated with copper sulphate or formalin after shelling and sown at Pusa, where groundnuts had not previously been grown, yielded a crop free from tikka, while we have had several cases where the parasite has been introduced with the crop when no precautions were taken to disinfect the seed. It is probable that it has come in, in these cases, in the form of spores in the soil adhering to the shell rather than in the seed itself, though in the United States, conidia have been found in centrifugal washings of the seeds.

---

\* It is probable that the fungus should really be considered one of the Tuberculariaceae and not a Septoglossum (Melanconiaceae) or Cercospora (Dematiaceae).

Treatment by spraying has not given good results in India. This is probably due to the difficulty of reaching the under surfaces of the leaves, which form a dense mat of foliage close to the soil.

Seed disinfection should be practised when the crop is grown at a distance from previously diseased localities, unless the seed is known to come from a place where the disease does not occur. Care should also be taken to remove the shells thoroughly and with them the adhering soil, before disinfection. Four hours in formalin in a strength of one pound to forty gallons of water, or half an hour in one-half per cent. copper sulphate solution, can be used.

Remarkable success in reviving groundnut cultivation in Bombay, where it had almost died out, has been achieved by the introduction of exotic varieties. The exports of groundnut from Bombay fell from 78,000 tons to less than 3,000 tons between 1894-95 and 1902-03. A number of exotic varieties were then imported from the United States, Japan, Mauritius, and Madras. These rapidly found favour and the exports rose to nearly 49,000 tons in 1912-13. The previous decline was due largely to the ravages of disease, chiefly tikka. The new varieties did not prove in any way immune to this disease, when first grown at Poona, but now that they are well-established, tikka has practically disappeared. Even when first introduced, they gave better results than the local sorts, since, though their leaves and stalks were freely attacked by the parasite, several of the varieties ripened much quicker than those previously grown and at the time of attack the nuts were already sufficiently developed to escape injury. This is one of the reasons why they were taken up so readily by the cultivators as practically to have replaced the old kinds.

The earlier trials showed that there was no inherent property in the tissues of the exotic varieties which enable them to resist the parasites when exposed to severe infection. But whereas the old varieties failed to give a remunerative yield when attacked by tikka, the new ones could be profitably cultivated even in face of it, and ultimately shook it off altogether. It seems difficult to account for this in any other way than by supposing that the old kinds had deteriorated by being cultivated too long under the local conditions (see p. 128). The groundnut is not indigenous to India, and the past history of the crop in this country, so far as it can be ascertained, indicates that previous failures have occurred and have been temporarily checked by the introduction of new seed. If this is so, we may expect to see another recrudescence of disease after a time, unless steps are taken to keep up a steady stream of new varieties.

There is evidence to show that with proper rotation and careful treatment, the crop on the Government farms does not rapidly deteriorate. How far this is permanent, or merely a postponement of an inevitable decline, is not yet known ; but it is a clear indication of the beneficial effects of good cultivation and especially rotation ; the latter, which is often neglected by the growers, is particularly necessary with this crop.

Another leaf spot caused by a species of *Cercospora* which seems to be as yet undescribed, is common on groundnut leaves in India. The spots resemble those of tikka disease, except in being less regular, lighter in colour, and not so sharply defined. The surface bears scattered, tiny, black points, instead of the larger, concentrically arranged, brown cushions of *Cercospora personata*.

The attack develops later than tikka and is not usually severe until after the nuts are formed. This, and the comparative absence of leaf shedding, account for the little harm caused by moderate attacks. Occasionally severe outbreaks have been seen, in which the number of spots was sufficient to cause the leaf to wither. In these cases, the leaflets were shed as soon as they had turned yellow, whereas in tikka the whole leaf often falls while still green except for a few spots.

Shade and excessive moisture are predisposing causes. In dry areas such as at Dhulia (Khandesh) there is much less than at Poona or in the eastern portion of the Central Provinces.

No treatment has been attempted for this last disease and, generally speaking, none is required.

**Root rot** (*Hypochnus Solani* Pril. & Del., *Rhizoctonia destruens* Tassi and *R. sp.*).—Groundnut is known to be attacked by the same two forms of *Rhizoctonia* in India as are found on the cowpea (p. 262) and also by *R. destruens* (p. 290). The disease is fairly common in the Bombay Presidency but is sporadic as a rule and not, like tikka disease, found throughout whole fields.

The general symptoms of the disease and the characters of the parasites have already been described under cowpea and potato. An important difference is that the underground nuts are sometimes attacked by the small, black *Rhizoctonia*, and the hyphæ may even penetrate into the embryo of the seed. Naturally, the position in which the nuts are developed renders them particularly liable to infection by soil-dwelling fungi.

It is probable, therefore, that some advantage may be gained by disinfecting the seed when known to come from contaminated soil. In the case of the potato, the use of formalin or corrosive sublimate has been found to reduce damage from *Rhizoctonia*, but here again rotation and good cultivation are the most important measures to adopt.

The small, black *Rhizoctonia* from cowpea and cotton is able to attack groundnut, but less virulently than the strain from groundnut itself; from jute, groundnut is, under normal conditions, not attacked at all.

### LINSEED (*Linum usitatissimum* L.).

**Rust** (*Melampsora Lini* (Lk.) Desm.).—This is one of the commonest crop rusts in Bihar and other districts where linseed is extensively grown. It occurs every year without fail at Pusa and is sometimes nearly as intense as a bad epidemic of wheat rust. At Pusa, flax has never been attacked, though grown for several years in the neighbourhood of severely infected linseed. As the two crops are merely cultivated races of the same plant, this indicates a degree of specialisation as great as that of the bean anthracnose (see p. 259). Flax was, however, badly attacked by rust in the Nilgiris a few years ago, and it was thought wise to destroy the crop (an experimental one) in order to prevent the flax strain of the parasite, which was doubtless introduced with the seed, from establishing itself in India. The rust is known on flax and on several other species of *Linum* in North and South America, Europe, Africa, Asia, and Australia. Each species seems to have its own specialised race.

Affected plants are very conspicuous owing to the bright orange-coloured pustules with which the leaves and stem are covered. At a later stage, as the crop ripens, the spots are mostly reddish-brown, turning to black, and are crust-like rather than pustular.

All four spore stages, spermatogonia, acidia, uredo, and telento, occur on the same plant, though the first two have, so far, only been recognised in the United States.

The spermatogonia have been found on rather youngish plants and probably only appear quite early in the season. They are small, inconspicuous, pale yellow, and of a flattened globoid or lens-shape. They are situated under the epidermis and may be in groups or scattered on both surfaces of the leaves. Their breadth is 100 to 175 $\mu$  and they are 85 to 95 $\mu$  high; the spores, which are borne on branched stalks (Fig. 133, 2), measure 3 to 4 by 2 to 3 $\mu$ , and there are no paraphysal filaments at the mouth.

The acidia are mostly on the under surface of the leaves, scattered, round, 200 to 400 $\mu$  in diameter, and bright orange-yellow in colour. They develop under the epidermis and burst out, surrounded by the torn tissues but without any peridium such as occurs in *Puccinia graminis*. This absence of the peridium is characteristic of what is known as the *Croma* type of acidium, common to all *Melampsoras*. The spores are in chains.

roundish, 21 to 28 by 19 to 27 $\mu$  in diameter, with a colourless, thin, warty wall and no distinct pores. They are formed by the division of a cell which arises from the fusion of two or more cells at the base of the aecidium in a kind of sexual process, the union of the nuclei being, however, delayed up to the teleuto stage (Fig. 133, 4—7).

The uredo sori arise from a mycelium derived in the early part of the season from the aecidiospores, later on from infection by the first-formed uredospores. They occur on both sides of the leaves, and on the stem and inflorescence. They are scattered or clustered, round on leaves, elongated on the stem, up to 1½ mm. across, powdery, prominent, and orange-coloured. The spores are roundish or ovate, 15 to 25 by 13 to 18 $\mu$  in diameter, with a colourless wall provided with fine warts and with indistinct, equatorial pores. Paraphyses are mingled with the spores and are large, strongly capitate, 40 to 65 $\mu$  long by 15 to 22 $\mu$  thick, and often curved (Fig. 133, 8).

The teleuto sori are reddish-brown, then black, shiny, often confluent, and form flat crusts on the leaves and stem. They often appear in the centre of a group of uredo sori. The spores are formed under the epidermis in a single layer of closely united, cylindrical cells, polygonal in section, and measuring 42 to 60 by 10 to 20 $\mu$  (usually about 55 by 10 $\mu$  in India) (Fig. 133, 9). At the base of the sorus are short, cubical cells, which lose their content as the spores develop. The germination is difficult to secure and is presumably of the usual type for the genus, by the production of a 4-celled promycelium from the end of the spore, each cell then bearing a small, round, yellowish sporidium.

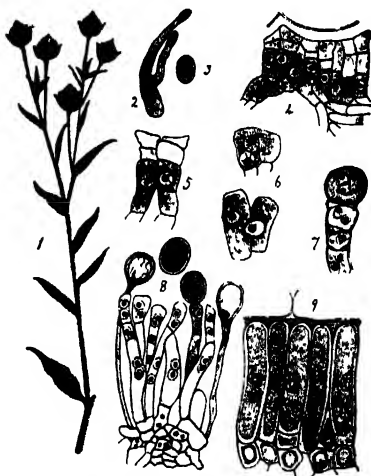


FIG. 133. Linseed rust (*Melampsora lini*): 1, part of plant with sori; 2, branched condiochore from spermatogonium,  $\times 750$ ; 3,  $\delta$  spermatium,  $\times 1125$ ; 4, young aecidium,  $\times 500$ ; 5, paired cells of same,  $\times 750$ ; 6, fusion of cells,  $\times 500$ ; 7, formation of aecidiospore,  $\times 375$ ; 8, part of uredo sorus,  $\times 375$ ; 9, part of teleuto sorus with fusion of nuclei,  $\times 375$ . (2—9 after Fromme).

Unlike the cereal rusts, the germinating teleutospores can infect, by means of their sporidia, the same host on which they were borne. Sowings on flax have given spermatogonia in 8 days and aecidia in 12. There is no reasonable doubt that this is the normal way in which the fungus is carried over to the new crop, and it also probably explains the reported conveyance of the disease by seed from Calcutta to Assam and Australia, since fragments of straw, bearing teleutospores, might easily find their way in with the seed.

The injury caused is considerable but is more difficult to estimate in linseed, where only a reduction in the amount of seed is caused, than in flax, where the fibre is rendered brittle and the damage is obvious. In the Central Provinces the rust appears in late November; in Bihar a

month or more later. In severe attacks, whole fields may be reddened by it and the crop ruined so as to be hardly worth the trouble and expense of harvesting. Such damage has not been observed at Pusa. At Dumraon, actual losses of over 28 per cent. by weight, in the seed of individual plants, have been recorded.

Direct treatment is no more likely to prove successful with this rust than with any of the others previously considered. There is, however, a decided possibility of checking infection by carefully destroying all the stubble and the debris from the threshing floors, since the teleutospores are not set free until the host tissues entirely disintegrate. If flax maintains its absolute immunity to the linseed rust in north India, it may be worth attempting to obtain immune crosses between the two, though there will probably be difficulty in getting a heavy seed yield with flax as a parent. This is, however, a matter for the Agricultural Departments and not for cultivators to consider.

### CASTOR (*Ricinus communis* L.).

**Seedling blight** (*Phytophthora parasitica* Dastur).—This parasite causes the most injurious of the fungus diseases of castor in India, destroying the seedlings and attacking the leaves of older plants. It has only been observed in the north-east up to date and is not known to occur outside India. The same fungus has, however, been found capable of infecting quite a number of plants, and it seems improbable that it is restricted to a single area.

The disease usually begins during the monsoon, when the plants are six to eight inches high. In low-lying, badly drained fields especially, 30 or 40 per cent. of the seedlings may damp off. The first indication of attack is the appearance of a roundish patch, of a dull green colour, on both surfaces of a primary leaf (cotyledon). This spreads to the point of attachment, causing the leaf to hang down in a rotting condition. Extension to the stem follows, and the seedling is killed either by the growing point being involved or by collapse of the stem. When true leaves of seedlings and the very young leaves of older plants are attacked, the disease similarly spreads down the petiole to the stem. The older leaves of large plants are also often attacked, but in this case the parasite remains localised on the leaf blade. Direct attack on the stem is only found in quite young seedlings; stems of plants three to six months old are immune from serious damage, though superficial injury occasionally occurs. This appears to be due to the structure of the tissues, the



cuticularised epidermis of older stems being able to resist penetration, and any entry through stomata being soon checked by the thick walls of the subjacent tissues. It is perhaps due to physiological causes that internal spread from the leaf blade to the petiole, and thence to the stem, does not occur, except in young plants or in very young leaves. The flowers and



FIG. 134. Castor seedling blight (*Phytophthora parasitica*) leaves with spots, showing both surfaces.

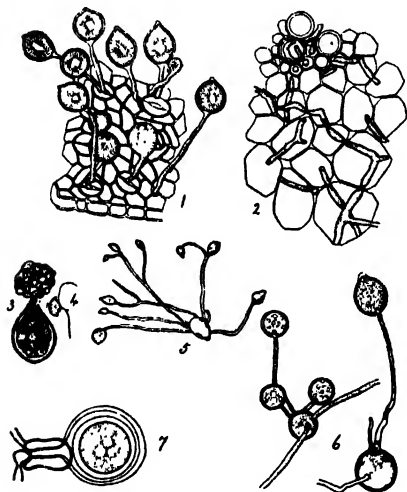
green fruits can be successfully inoculated in the laboratory but have not been found attacked in nature, as the fungus has ordinarily ceased its activity at the time of flowering. Except in seedlings, the leaf attack does little damage to the crop when grown for seed. Of late years, however, castor has become somewhat extensively cultivated as a food for

the *Eri-silkworm*. The leaf disease is more harmful in this case, as the worms do not thrive when fed with diseased leaves.

The leaf spots rapidly turn yellow and then brown. As centrifugal spread goes on, concentric zones of darker and lighter brown are found. The outer border is ill-defined, greenish above, brownish-grey below. Two or more spots may coalesce, often covering almost the whole of the leaf. The veins offer little resistance to spread, even in mature leaves. Diseased leaves tend to fall prematurely.

On the under surface of the leaf spots and also in moist weather on affected stems, a very fine, whitish haze, hardly visible without a lens, can be found. This is the fructifying stage of the fungus.

The mycelium within the tissues is both intra- and intercellular (Fig 135, 2).



1: 1, surface of leaf as in tissues of stem,  $\times 100$ ; 2, a sporangium discharging,  $\times 200$ ; 3, a zoospore,  $\times 320$ ; 4, a sporangium germinating by germ-tubes and secondary sporangia,  $\times 50$ ; 5, chlamydospores, one germinating and forming a secondary sporangium,  $\times 50$ ; 6, zoospore showing antheridium encircling the stalk,  $\times 580$ .

Haustoria are scarce and it is even open to doubt whether the haustorium-like bodies found within the cells are anything else than young intracellular hyphae. All the leaf tissues are invaded, even the fibro-vascular bundles, the hyphae entering thick-walled cells and vessels through the unthickened gaps. After a few days' growth within the tissues, numerous branches penetrate the lower epidermis to reach the surface and become sporophores. These emerge usually through the stomata or between two adjacent cells, less often by breaking through an epidermal cell. They come out singly or sometimes in twos or threes, never in larger clusters.

The sporophores are slender, unbranched, and of variable length, from 35 to over  $500\mu$  (usually from 100 to  $300\mu$ ). At the tip of each is a single, colourless, ovoid or roundish

sporangium (Fig. 135, 1), measuring from 25 to 50 by 20 to  $40\mu$  in diameter, which falls off when ripe without any stalk. A small but distinct papilla is found opposite the point of attachment to the stalk, and the ripe sporangium, when sown in water,

liberates zoospores through the opening formed by the rupture of the wall at this spot (Fig. 135, 3). The number of zoospores formed varies from 5 to 45 (average about 30). In artificial culture, the sporangia vary within wider limits than on the leaf and their stalks are sometimes irregularly branched. Lateral and intercalary sporangia (the latter usually round) are also found in culture.

The zoospores are of the ordinary *Phytophthora* type (Fig. 135, 4). Their discharge is more readily obtained at a moderate than at a very high temperature. Light influences the discharge favourably, while darkness inhibits it. Cultures kept in the dark from the beginning, form no sporangia until brought into the light. Moisture is equally necessary for the production of sporangia, which fail to develop on inoculated plants kept in a dry room. The zoospores come to rest in from 20 minutes to two hours after discharge, and germinate about 15 minutes later by one, or rarely two, germ-tubes.

As in other allied species, many sporangia, especially old ones and those not sown in pure water, germinate directly by one or more germ-tubes instead of liberating zoospores (Fig. 135, 5). These germ-tubes may form a mycelium or develop secondary sporangia, which again may germinate directly or may form zoospores.

In cultures and on inoculated castor fruits, a second spore form, chlamydospores similar to those described in *Phytophthora Colocasiae*, is produced. They are round, thick-walled, yellow bodies, 20 to 80 $\mu$  in diameter, and formed either at the end or in the course of a hypha (Fig. 135, 6). Extremes of temperature, such as retard the formation of sporangia, do not affect the production of these spores, which are freely formed in the hot, dry months when sporangia are scanty. They retain the power of germination for many months after the sporangia and hyphae are dead. They give out one or more germ-tubes, which may form a small mycelium, or sporangia, or new chlamydospores.

Oospores (Fig. 135, 7) have been obtained in artificial cultures of the fungus on certain nutrient substances but, as in the case of the two allied species which cause potato and *Colocasia* blight, they have not, so far, been discovered in diseased plants, or anywhere else but in the laboratory. They agree in their general characters and mode of formation with those of the two species referred to. The antheridium is first formed as a swelling at the end of a hypha or in its middle. After this is cut off by one or two septa according to its position, the branch which is to form the oogonium grows through it and out on the other side, immediately swelling up as it emerges into a nearly round cell, cut off at the base by a septum. This is the oogonium. The oospore now forms within the oogonium, nearly, but not completely, filling it. The oogonium measures 15 to 27 $\mu$  in diameter (average 23.8 $\mu$ ) and has a thick, yellow wall. The oospore varies from 13 to 24 $\mu$  (average 18.6 $\mu$ ) and is round and colourless. At the base of the oogonium, the antheridium remains attached for a long time. These spores have not been germinated, but it is certain that they preserve their vitality for long periods. Like the chlamydospores, it is probable that, if formed in nature, they serve the purpose of carrying the parasite over from one crop to the next.

Experiments indicate that the fungus remains in some form in the soil of diseased fields for at least several weeks. Seedlings sown in soil a month after the previous crop had been killed off, were attacked at the soil level and collapsed. After two months, no attack occurred. The regular recurrence of the disease at a certain period each year, some nine months after the last trace of the previous attack has disappeared, suggests that the infective matter does, however, persist in the soil, perhaps in very small quantity. A single germinating chlamydospore would be enough to start the disease if it reached a leaf or young stem,

and subsequent spread would readily result from the first-formed sporangia, provided the weather conditions were suitable; the sporangia are light enough to be freely disseminated by the wind, and in damp weather they would germinate readily on the leaves. Inoculation experiments prove that the germ-tubes of the zoospores enter rapidly into the leaf, both through the stomata and directly through the epidermal cells. The new spot of disease is visible within 24 hours and new sporangia are formed in two days.

This fungus has been proved capable of infecting a number of plants of different kinds, when artificially inoculated. Amongst these are young potato, tomato, and brinjal plants, and seedlings of several garden annuals. It has been found also causing a leaf disease of til (*sesamum*) and it, or a closely allied species, attacks the flowers of *Sida cordifolia*, a wild, fibre-producing plant.

This disease would probably not repay direct treatment by spraying,

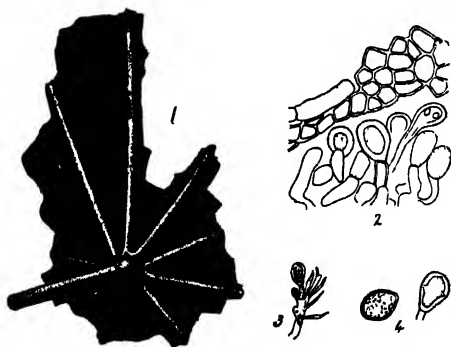


FIG. 136. Castor rust (*Melampsorella Ricini*): 1, under surface of leaf with sori,  $\times \frac{1}{2}$ ; 2, part of young uredo sorus,  $\times 430$ ; 3, spore stalk,  $\times 200$ ; 4, uredospores (thin and thick walled),  $\times 200$ . (2-4 after Ajrekar).

as it is not nearly so virulent as potato blight or even *Colocasia* blight. It is only in damp, low-lying localities that it seems to be able to do damage and the sole recommendation that can be made at present in regard to it is to avoid growing the crop in such situations.

**Rust** (*Melampsorella Ricini* (Biv.-Bern.) de Toni).—The rust of this crop is very common in the southern half of the Bombay Presidency and also occurs in the Central Provinces. It is known in southern Europe, north, east, and south Africa, and Ceylon, but always, as in India, in the

uredo stage only. The complete life-history is, therefore, unknown and it is doubtful that it belongs to the genus *Melampsorella*.

The disease usually appears in Bombay between November and February, on castor sown in June as an annual crop, reaching its height about January. It has been found in September near Poona on old plants, and in this case had probably persisted through the hot weather. In severe attacks, practically every leaf is thickly covered with rust pustules, the effect being to cause them to dry up and wither prematurely.

The uredo sori are borne chiefly on the under surface of the leaf, rarely on the upper surface. They are often grouped in small circles, and two or more frequently coalesce. They are at first covered by the epidermis and a layer of fungal cells, but become exposed later on, being then powdery, raised, and orange-yellow in colour. The upper surface of the leaf is marked by corresponding small, roundish, yellow spots.

The hyphae within the leaf collect in masses below the epidermis, forming a minute cushion. The layer next under the epidermis is formed of polygonal cells, derived from specialised hyphae. Below this, the spores are produced on curiously branched hyphae, only some of the branches bearing uredospores at their tips, the others being sterile. Stout, club-shaped paraphyses are also intermixed with the spores. As the sorus matures, the epidermis and "peridium" (or covering of fungal cells) are ruptured and the spores set free to the air.

The uredospores are round or elliptical, finely warty, and with orange contents. In fresh sori, two kinds may often be distinguished, one with very thick and the other with thinner walls. In old, dried sori, the proportionate number of thick-walled spores increases. Both sorts agree in other respects and germinate similarly. Their size varies from 25 to 29 by 19 to 25 $\mu$ . On germination, several germ-tubes emerge through the pores in the wall (which are up to 6 in number) but only one usually continues to grow. The uredospores are capable of infecting the castor leaf and producing fresh uredo sori, but the further life-history, if any, is unknown.

No suggestions for treatment can be given, beyond removing the leaves which are first attacked, in order to check spore dissemination.

**Leaf spot** (*Cercosporina ricinella* (Sacc. & Berl.) Speg.).—This is very common at Pusa and doubtless in other parts of India, causing much injury to leaves intended as food for the *Eri*-silkworm, but otherwise doing little damage to the plant. It is known also in North America, Australia, Ceylon and China.

The leaf spots are visible on both surfaces, at first as minute, black or brown points, which soon become surrounded by a pale green ring. As they enlarge, the centre turns pale brown and then greyish-white and the margin forms a deep brown band, which may be narrow and sharp or broad and diffused. In the white centre, tiny, sharp, black dots may be seen, the conidiophore clusters of the fungus.

The spots occur often in great numbers, scattered over the leaf. They are roundish when young but may become irregularly angular when mature, and are often checked in growth by the main veins of the leaf.

They vary in size from  $\frac{1}{2}$  to 3 mm., or rarely more. When many are close together, the intervening leaf-tissue withers and large brown patches of



FIG 137. Castor leaf spot (*Cercosporina ricinella*) leaf showing spots,  $\times \frac{1}{2}$ ; conidiophores and conidia,  $\times 200$ .

straight or slightly curved, and divided by several (up to 7) transverse septa. They are up to  $105\mu$  long by 4 to  $6.5\mu$  broad.

The damage done by this fungus is practically confined to crops grown for *Eri*-silkworm food, the insects disliking the spotted leaves and suffering when fed on them. As leaves for this purpose cannot be sprayed, the only hopeful way of attempting to check the damage would seem to be the use of immune or resistant varieties, if any such exist or can be produced. No experiments in this direction appear to have been attempted.

dried leaf may result; but, even in this case, the individual spots can be made out by their deeper brown margin.

The growth of the mycelium in the leaf is strictly limited and ceases when reproduction begins. The hyphae collect to form very small stromata under the epidermis, through any part of which, not alone through the stomata as occurs in many allied fungi, the clusters of conidiophores emerge.

The conidiophores are in groups of from 10 to 20, clear brown below, lighter towards the tip, septate several times, unbranched, and measure from 24 to 70 by 3 to  $6.5\mu$ . The upper part is usually knobly or flexed on account of further growth taking place below each conidium, as is usual in the genus.

The conidia are elongated, almost colourless bodies, tapering above and truncated below,

## CHAPTER IX.

### DYE, DRUG, AND SPICE CROPS.

#### **TOBACCO** (*Nicotiana Tabacum* L. and *N. rustica* L.).

**Bacterial wilt** (*Bacillus solanacearum* Smith).—This disease occurs annually in the Rangpur district, Bengal, one of the most important centres of tobacco cultivation in that Province. It has been known for many years under the local name of "rasa," signifying moisture, the popular idea attributing it to excess of soil moisture. There is no information as to its occurrence in other parts of India, but it is reasonable to suppose that it exists elsewhere, especially as the potato disease caused by the same bacillus appears to be fairly widely distributed (see p. 274). Outside India, it is known in the United States, Japan, Sumatra, Java and probably elsewhere.

The distribution of the disease in the infected fields is irregular and patchy. The cultivators, as is so often the case, express but little anxiety over its occurrence, regarding it as an inevitable dispensation of Providence; this is, no doubt, due to the generally low average of cases, which seldom amount to more than 5 or 6 per cent., though some fields with 20 to 25 per cent. of affected plants may be seen. As the disease has not been under observation for many years, it is, of course, possible that more severe outbreaks may occur, like those known in parts of the United States and, especially, in Sumatra.

The symptoms resemble those of other wilt diseases, the base of the stem and the main roots being discoloured and partly rotted, while the green parts (leaves and green shoot) undergo a progressive withering. On splitting the stem, the brown colour of the tissues is found to extend from the surface inwards to the pith, and to spread from the collar (probably the usual point of infection) up the stem and into the leaves. It is visible externally as dark brown streaks on the stem, and midribs of the leaves. Shrinkage of the tissues in the neighbourhood of the point of attack has been noticed in some cases, particularly in artificial inoculations above the ground level. The harder tissues at the ground level would naturally show this less, where the infection occurs at that point.

Similar shrinkage may be seen sometimes in the tissues of the leaf bases, when the leaves wither, and also in the young shoot poisoned by the toxins produced by the parasite

On microscopic examination, marked necrosis and disintegration of the tissues of the diseased parts is found. The middle lamella of the dividing walls between adjacent



FIG 138 Bacterial wilt of tobacco (*Bacillus solanacearum*) wilting of plant artificially inoculated at A

cells is dissolved and the cells consequently separate from one another. The cells themselves, particularly near the point of infection, are filled with masses of bacterial slime, which also oozes out of the out ends of the vessels. This slime contains a large variety of bacteria, as numerous saprophytic organisms follow in the wake of the true parasite. It is only in the earlier stages that *B. solanacearum* predominates. In artificial inoculations, the bacteria are at first confined to the particular set of water vessels into



which the original inoculation took place, and the leaves occurring vertically above this point are the first to show symptoms of wilting. Lateral spread takes place later on.

The symptoms appear to be due more to the action of toxins secreted by the parasite in destroying the cell protoplasm and walls, than to any direct mechanical interference with the water supply, owing to the vessels being stopped by bacterial masses. The

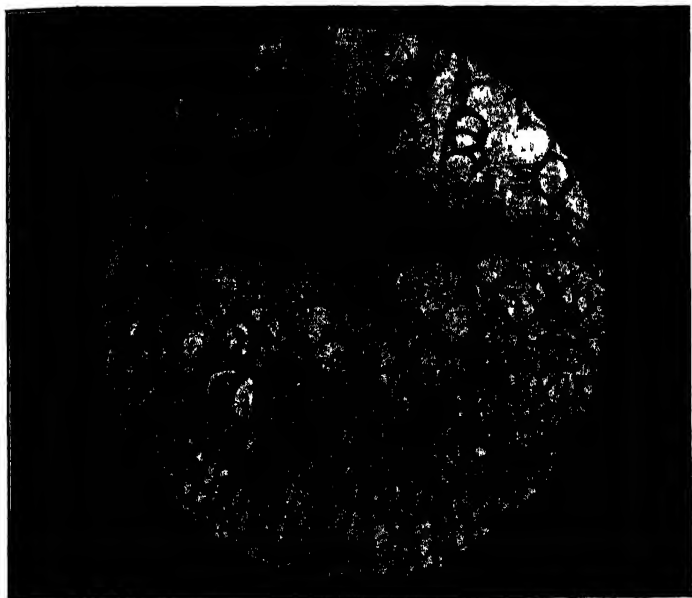


FIG. 139. Bacterial wilt of tobacco (*Bacillus solanacearum*). necrosis of tissues and separation of cells by solution of the middle lamella.

microscope shows that this plugging is not sufficiently pronounced to cause wilting, and experiments intended to cause similar symptoms by cutting the vessels and introducing plasticine (moulding wax) failed. On the other hand, wilting and disintegration of the tissues can be caused by the injection of a solution of toxins from cultures of the bacillus, freed from living bacteria. Death of the cell protoplasm, of course, causes the tissues to wither, and the water supply is interfered with by the cessation of osmotic action. At a later stage, no doubt, the water supply is further checked by the formation and accumulation of gum masses in the vessels. Such gum masses are a prominent feature of the disease (Fig. 43).

The parasite is a minute, rod-shaped or oblong bacillus, from 1 to  $1.5\mu$  in length by  $0.6\mu$  in breadth. No flagella have been observed and the motility is doubtful in the Indian strains. The cultural characters, which in the group of plants to which this

organism belongs are of great importance in recognising the identity of species, are as follows :—

*Agar slant*.—Growth white moist smooth becoming sepia brown ; agar stained.

*Gelatine*.—Colonies round white thin ; opalescent ; brown under magnification, by transmitted light. No liquefaction.

*Bouillon*.—Turbidity on shaking ; alkaline reaction.

*Potato*.—Growth watery and colourless at first, then opaque white, becoming brown and finally bitumen black.

*Glucose bouillon*.—No acid or gas.

Pure cultures of this organism, which quite agrees with *B. solanacearum* Smith, except in the absence of flagella and motility, produce the disease when inoculated into healthy tobacco plants. Potato cultures produce obvious symptoms in from 5 to 8 days when inoculated by a single needle prick into the stem, whereas agar cultures require from 8 to 12 days, and those in bouillon seldom show results under 18 days. Strains whose virulence has been reduced by culture on artificial media sometimes require as much as 5 weeks. When the dark brown pigment found in the later stages of culture on potato and agar has appeared, the virulence is lost.

In the field, the infecting bacillus is probably unable to gain entrance into the plant, except through the wounds caused either by some mechanical agency or by soil nematodes or insects. The operations of transplanting and interculture and the practice of removing the lower leaves, no doubt afford numerous channels of entry into the base of the stem or upper roots. Attempts to produce the disease by watering the soil with cultures of the parasite failed, except when previous injury to the roots had taken place. Recently transplanted plants can be infected in this way, owing to the disturbance of the root system and lowered vitality consequent on the operation. No connection has been traced between the soil conditions and incidence of the disease, beyond the general lack of good root development in diseased plants. So far from excess of moisture being the cause of the disease as supposed by the cultivators, it is probable that lack of moisture during the long rainless period of the cold weather in Bengal reduces the vitality of the plant and increases the channels of infection by accelerating the casting of the lower leaves, before they have been cut off at the base by the usual protecting layer of cork cells. As a matter of fact, the attempts of the cultivators to dry the soil by repeated shallow ploughings and hand hoeings, actually have the opposite effect, in tending to conserve the soil moisture by the formation of a surface mulch.

The treatment depends more on the adoption of suitable methods of cultivation than on any direct attack on the bacillus. Field practice should aim at conservation of the soil moisture and development of the root system, so as to produce better grown and, consequently, more resistant plants. It has been found also that the parasite does not

survive a temperature of 50°C. for 30 to 40 minutes, and the intense heat of the sun in India, at certain times of the year, renders it possible to make use of the bactericidal effect of such high temperatures.

Efficient hot weather cultivation is, therefore, of importance in several ways. It will help to destroy a large percentage of those bacteria that remain in the surface soil, and in diseased and dried up tissues; the exposure of the surface soil to high temperatures in the hot weather in Bengal is, no doubt, largely responsible for the absence of accumulation of this disease which has been noticed at Rangpur. Hot weather cultivation also results in securing a better water supply in the soil during the following cold weather; the root development is consequently encouraged and the vigour and vitality of the plant increased. The methods of the best Rangpur cultivators should be followed and extended to affect a deeper soil stratum, so that in addition to maintaining a loose surface mulch during the growth period, increased root development may also be secured. Particular attention should be paid to drainage, as this is of vital importance in producing a vigorous root development.

Transplanting should be done early, while the plants are still small; in this way there is less risk of injury to the plant either by loss of roots or leaves, fewer channels of entry for the parasite are formed, and the lowering of resistance of the plant to attack lessened.

If nematodes or other soil organisms should be found injuring the roots, the adoption of some measures calculated to check their activity may be required. Trap cropping for nematodes, such as is employed in America, where such plants as cowpeas are first grown and, when well infested with nematodes, pulled out and destroyed, might be tried.

It has been found in the United States that the use of mineral fertilisers which tend to produce an alkaline reaction in the soil, such as carbonate of potash, lime, and ashes, has a decidedly prejudicial effect on the root growth of tobacco; such a condition would almost certainly conduce to the multiplication of *B. solanacearum* (which produces an alkaline reaction in the tissues infected by it and, therefore, presumably prefers an alkaline condition), while at the same time weakening the plant in its resistance to attack. This aspect must be borne in mind, if the use of such fertilisers becomes common in India.

The length of time the bacillus can live in the soil is not known. In the United States, rotation greatly reduces the disease, provided a susceptible crop is not grown oftener than once in five years.

In Sumatra the bacillus is said to remain for eight years or more, in the absence of tobacco.

No variety of tobacco has yet been found to resist the disease, though very extensive tests have been carried out in Sumatra. Attempts are now being made in that country to obtain resistant forms by selection and crossing.

Besides tobacco and potato, the same parasite is known, outside India, to attack a good many other plants, some, such as tomato, brinjal, and dhatura, belonging to the same family, others not related. In all it has been found on 15 species, belonging to 5 families, in Sumatra. Groundnut Java indigo, chillies, and til are amongst these, and in India may be expected to suffer from at least an occasional attack.

A tobacco disease of the "wilt" type sometimes does a good deal of damage in Gujarat, but it has not yet been settled whether it is due to the above cause or to a *Fusarium*.

**Mildew (*Erysiphe Cichoracearum* DC.).**—The powdery mildew of this crop is not common in Bihar, but occurs fairly often at Rangpur (Bengal) under certain conditions, and also in parts of Madras. It is known in south Europe, Ceylon, the Dutch Indies, Australia, and South Africa, though, as the perithecial stage is rarely formed, there is some doubt whether these are all due to the same species.



FIG. 140. Tobacco mildew (*Erysiphe Cichoracearum*): leaf with mildew,  $\times \frac{1}{2}$ .

The mildew forms a fine whitish web on the leaves, chiefly on the upper surface but also underneath; this becomes powdery when spores are formed. At first the patches may be small and isolated, but they soon join and may cover a good part of the leaf. In the neighbourhood of the larger veins, the covering is usually thicker than elsewhere.

Affected leaves wither and turn brown, at first in the densely covered parts, later over the rest of the surface. The tissues dry up and are easily broken. Usually the lower leaves are first attacked, the disease appearing at a time when the crop is sufficiently grown to afford shade to the lower leaves. Later, the middle and upper leaves may be affected, and in shady localities

or where the crop is very dense, the spread may be rapid. Seedlings are reported to be sometimes attacked in other countries.

The parasite is confined to the surface of the leaves, sending only suckers (haustoria) into the epidermal cells. The mycelium forms a fine or dense network according to the vigour of its development, composed of branching hyphae, from which upright chains of conidia, of the customary oidial type found in the mildews, arise. The chains consist of several cylindrical or barrel-shaped spores, 26 to 35 $\mu$  long by 15 to 20 $\mu$  broad, rounded at the ends, and hyaline. They are readily detached and germinate in water or very moist air, forming usually a single germ-tube. These spores do not preserve their vitality for long, and cannot, by themselves, carry over the disease from one season to the next.

At a later stage, the perithecial spore-cases, which alone, in this group of fungi, permit of the ready identification of species, have been found in south Europe on rare occasions. They prove the fungus to be the same as that already described as causing the mildew of bhindi (p. 306) and *Cephalandra* (p. 314). This stage of the parasite has not yet been discovered in India or Ceylon. It consists of minute, black dots, found partly immersed in the white patches and scarcely visible to the unaided eye. Each dot consists of a single spherical perithecium, agreeing with those already described on p. 306. The asci are usually about 10 in number and contain, as a rule, two spores each.

The development of the fungus depends largely on the humidity of the air around the leaves, and a suitable range of temperature. In Rangpur, the disease has only done damage in heavy crops in shady localities. In other countries, badly drained fields have been reported to suffer considerable damage in wet seasons.

The treatment should be directed, in the first place, to securing proper aeration of the plants by wide spacing and the avoidance of shade. Nothing seems to be more effective in checking the spread of the fungus than direct exposure to sunlight, and for this purpose planting in properly spaced rows is important. Excessive moisture during the tobacco season is less to be feared in India than in most other countries, and the absence of the disease in Bihar is probably largely due to the dryness of the tobacco fields during the cold weather. In other localities it may be necessary to avoid low-lying fields and to attend to drainage. Clean cultivation is especially necessary where this disease is prevalent, both because the fungus is known to be able to attack various weeds and may, perhaps, be carried over from season to season on these, and because clean cultivation promotes aeration and keeps the surface soil dry.

The use of sulphur, so valuable in allied diseases, is difficult in tobacco, as the leaves may still retain some of the fungicide when plucked, and the quality of the tobacco be ruined. In Italy, it is sometimes dusted on the lower leaves at the time of first appearance of the disease and these leaves, being considered of little value, are not harvested. As the disease

almost always begins from below, good results are said to have been obtained by this practice. In India, hand picking of all infected leaves at the time of removing the lower ones would probably be preferable, but care should be taken to place them at once in a bag or something of the kind, so as to avoid scattering the spores on non-infected leaves. The picking should be done in the morning on dry, sunny days, so that the disseminated spores may have less chance of germinating. There would be no objection to sulphuring the seedlings, should the disease appear in that stage.

There is some evidence that certain varieties of tobacco are less susceptible than others, this being the case with Virginia and Kentucky varieties grown in Italy. It is not known whether this applies to any of the varieties found in India.

A disease known as "Kapti" in the Azamgarh district of the United Provinces is, from the description, very possibly identical with the above.

**Leaf spot** (*Cercospora Nicotianæ* Ell. & Ever.).—This disease is quite common in India and occurs also in almost all tobacco-growing lands, though apparently rare in Europe. Beyond injuring leaves intended as wrappers for cheroots, it does not seem usually to do much damage. Even the wrappers are not injured if the spots are not too numerous and the dried tissue does not crack and fall out. In some places such leaves are prized as marking certain brands of cigars.

Small, roundish spots, about  $\frac{1}{4}$  to 2 cm. across, appear on the older leaves. Usually spread is limited, as with most parasites of this genus, but sometimes, especially at the tip, several may combine and large, irregular patches result. The spots are brown, becoming gradually white in the centre and drying up. In Sumatra, two distinct types of disease have been described, one causing reddish-yellow speckles and the other black spots. The fungus occurs on both and one can be produced by inoculating from the other.

The mycelium is found between the cells, which are rapidly killed. Small clusters of hyphae collect in the spaces below the stomata and send bundles of conidiophores through the opening. They are most numerous on the under surface.

The conidiophores are unbranched, greenish-brown, up to 250 $\mu$  long by 4 to 6 $\mu$  broad, with 1 or more septa, and bent in the manner usually found in the genus.

The conidia are straight or slightly curved, very long in proportion to their breadth almost colourless, with from 2 to 12 septa, and 50 to 180 by 4 $\mu$  in diameter.

The species in the Dutch Indies was at one time considered distinct from that in the United States, but the Indian and Ceylon forms are so variable that probably all belong to the one fungus.

Overcrowding and excessive moisture within the crop are considered in the Philippines to increase the severity of the disease, while open planting and the early removal of the lower leaves diminish it. When the leaf cracks or dries from too many spots, the damage may be



FIG. 141. Leaf spot of tobacco (*Cercospora Nicotiana*): 1, part of young leaf with spots,  $\times \frac{1}{4}$ ; 2, spots on mature leaf,  $\times \frac{1}{4}$ ; 3, conidiophores and conidia,  $\times 200$ ; 4, conidia germinating on leaf and germ-tubes entering stomata. (4 after Sturgis).

serious. In tobacco for native consumption in India, the effect is insignificant and no treatment is required.

**Mosaic disease.**—By this name is known a peculiar disease of tobacco found apparently in every tobacco-growing country of the world, and in the sum total of its effects undoubtedly the cause of greater losses than any other disease of the crop. It is, perhaps, the best known example of a group of diseases of obscure origin, in which not only has no parasite been detected, but there is strong reason for believing that none of any type known to us can exist. Some hold that it is a physiological disorder, due to an innate alteration in the processes or functions of the plant, not brought about by the presence of an independently living parasitic organism; others, that it must be parasitic but due to a form smaller than any yet known, or even to a "living contagious fluid"—a suggestion which goes still further beyond the limit of any life known to us. It seems more reasonable to suppose, as other investigators have,

that the virus is one of an enzymic nature, capable of producing extensive effects in minimum doses. In the United States, it has been claimed that the direct cause is an excessive quantity of an oxidising enzyme present in the cells, and that it is possible to produce the condition artificially without any infection from outside. Whatever the cause, it is probable that it is not peculiar to tobacco. Similar diseases are known in tomatoes, chillies, and other plants, some being identical with that of tobacco, others distinct. They form a group which is perhaps the most obscure in the whole field of vegetable pathology.

Mosaic disease has not been especially studied in India, and all that is known of it in this country is that it is prevalent in many tobacco tracts. The chief loss is said to be in tobacco intended to be cured for European consumption; that grown for indigenous use is less depreciated in value, since the reduction of elasticity, poorness of burning quality, and inferiority of aroma, brought about by the disease, are not of consequence. The natives of Sumatra are even said sometimes to prefer the diseased leaves. Still the loss in yield is enough to be of significance, no matter for what purpose the tobacco is grown.

The most characteristic symptom is a patch-work appearance of lighter and darker green areas on the leaf. The lighter green patches may, in severe cases, become almost white. They lie between the lateral veins, seldom reaching quite up to the latter. The intervening portions may be of normal green or of a deeper colour than normal, and are often oblong and so placed that they are crossed in the direction of their longer axis by a vein. The boundary between the patches is sharp, but along the finer veins a dark green area may be prolonged a little way into the lighter green, causing an irregular outline. The severity of the attack is marked by the extent to which the lighter green areas cover the surface. It is most visible on the younger leaves, especially at the top of the main stem and in the small side shoots. The latter usually show it strongly when they arise in the axil of a diseased leaf.

The darker green areas in the leaf are thicker than normal, due to an increase in the palisade tissue, and are often bulged on the upper surface and hollowed below, owing to uneven growth. In the pale areas, on the contrary, the mesophyll may be feebly developed. This alternation of thinner and thicker portions during growth often leads to distortion, curling, or crumpling of the full-grown leaf. Occasionally the actual size of the leaves is much diminished. Other deformities sometimes found on the diseased leaves (such as foliar outgrowths) seem to be accidental, as they occur also on plants not suffering from mosaic.

In the final stage, the chlorophyll in the lighter areas is mostly destroyed and the cells dry up and collapse. But this is often delayed until the time for harvest has come, and there is not much actual loss from it.



The symptoms may begin at any stage in the growth of the plant, even in seedlings before transplanting. Whenever they appear, the already opened leaves remain unaffected and only those newly formed show them. This is why the growing top and young lateral shoots frequently exhibit the presence of mosaic best.

The disease is highly contagious. Inoculation with a tiny drop of expressed sap from a diseased leaf, large enough only to be carried on the point of a needle, causes the disease without any loss of virulence, even when diluted with 1,000 parts of water. Dilution to 10,000 shows some attenuation, but still often causes the disease. It has long been known that filtering the sap through the usual germ-proof candle filter does not affect its virulence. It is actually said, and supported by very strong evidence, that coolies working in the fields at topping, removing leaves, or picking off the grubs that damage the crop, can convey the disease by merely touching sound plants after a diseased one. In such cases, it appears as if the application of the virus to the unbroken leaf surface is sufficient. The young parts of the plant are the most highly infectious.

The incubation period between the time of infection and the appearance of external symptoms is one or two weeks, usually about 9 to 12 days. Probably spread is limited, as actual contact seems to be required, except where the virus is carried by insects or by man. It is said also that the roots may be infected from diseased material in the soil. Seed from diseased plants may give healthy or diseased progeny, and there is no evidence that mosaic is hereditary, though a predisposition to it may be. In virgin soil it is usually absent for some years.

There seems to be no way of preventing this disease, and all that can be done is to attempt to lessen the liability to it and to reduce its conveyance by the field workers. Seed from healthy plants should be used.

The danger of starting infection from plant debris in the soil, from a previous crop, should be borne in mind, especially in selecting the seed bed. When topping or handling of any other sort has to be done, diseased plants should be left alone, to be gone over separately afterwards.

The species known as *Nicotiana viscosum*, and some of its hybrids with *N. Tabacum*, are immune to the ordinary form of mosaic in the United States, but are affected by a distinct form of the disease. *N. rustica* is said to be immune in Europe, but is very severely attacked in the United States. There is thus scope for work in attempting to breed resistant races.

**OPIUM** (*Papaver somniferum* L.).

**Downy mildew** (*Peronospora arborescens* (Berk.) de B.).—The downy mildew of the opium poppy occurs in a mild form throughout the chief opium-growing tract in Bihar and the east of the United Provinces every year. In many years, there are localised attacks of greater severity, and occasionally epidemic outbreaks have affected considerable areas. It was believed till recently that the enormous losses caused by "blight" in the past, the history of which is found in Government Records from time to time, as in 1871 and 1874, were due to this disease; but the investigation of the serious outbreak in 1913 showed that part of the damage was due to *Rhizoctonia*, and some of the old descriptions, though imperfect, agree in the main with the disease caused by the latter fungus.

The importance of the crop in Indian agriculture has been much reduced by the measures now in force to restrict the use of the drug. It has disappeared from the neighbourhood of Pusa, and the investigation of its diseases has naturally suffered. Enough has been ascertained to show that the fungal diseases of opium rank among the major calamities which have befallen the Indian cultivators in certain tracts, and that the losses of 20 to 30 per cent. of the yield, amounting to between one quarter and half a million sterling in value to the cultivator, which have been recorded in years such as 1871, may be traced with considerable probability to the present disease and that next to be described.

The first appearance of the mildew is usually about February, near Pusa, when the plants are almost fully grown. In other countries, where wild and garden poppies are infected by the same fungus, it is said to appear from the seedling stage, but this has not been observed at Pusa though some of the severe outbreaks in the past have been described as beginning in November or December. At the time of flowering, diseased spots may appear on the inflorescence, but usually the chief damage is to the leaves. These are covered with pale brown spots, chiefly collected near the tip and margin. In favourable weather conditions, these spread, the whole leaf may be involved, and extension to the stem may occur. The attacked areas dry up and wither, the tissues becoming brittle and easily broken. In bad attacks, the leaves wither in their entirety. On the under surface is found a very fine, grey-violet down, scarcely visible, formed by the conidial stage of the parasite.

The mycelium of the fungus ramifies between the leaf cells, sending short, rounded haustoria into their interior, and gradually killing them. Hyphae extend to the spaces beneath the lower stomata, and send conidiophores singly, or in little groups of five or six through the orifices to the surface.



Fig. 142. DOWNY MILDEW OF OPIUM POPPY



The conidiophores are robust, erect, and very long, sometimes nearly 1 mm. in length, by 10 to 12 $\mu$  thick. They are 7 to 10 times dichotomously branched towards the top, the ultimate branches being very fine, curved, short, and diverging nearly at right angles. On the pointed tip of each there is a single round-oval conidium, hyaline or very pale violet in colour, and measuring 20 to 25 by 18 to 22 $\mu$  in diameter. The conidia germinate by a germ-tube either from the end or laterally.

In the older withered spots, oospores are developed within the tissues, often in quantity. The oospore is surrounded by an irregularly thickened, reddish-brown wall, formed of periplasm and 4 or 5 $\mu$  thick in places, outside which the thin oogonial wall lies. The oogonium averages 33 $\mu$  in diameter, the oospore being 21 to 32 $\mu$ , round, and of a yellowish colour.

As is normal in this genus, the conidia are short-lived and the disease is carried over by the more resistant oospores from season to season.

In the moist climate of lower Bengal, the ornamental varieties of *Papaver somniferum* suffer so much from this mildew, that as a rule they are not grown in the Calcutta Botanic Gardens. The still more common garden forms of *Papaver Rhoeas* are said, on the other hand, to be little attacked. Garden poppies are not attacked in Pusa, but the allied weed *Argemone mexicana* (vern. *kateya*), not uncommon in Bihar, is frequently infected and should therefore be weeded out from the vicinity of the fields.

There seems to be no record of attempts to control the disease by spraying. Bordeaux mixture would probably give good results, but as the financial conditions under which the crop is grown are peculiar, the produce being a Government monopoly, careful experiments would be required to determine whether spraying would repay its cost to the cultivator.

All withered leaves and debris from the crop should be carefully collected and burnt, in order to prevent the reappearance of the disease the following year, through oospores set free by the disintegration of diseased tissues in the soil.

Recent work has shown that different cultivated races of the opium poppy vary in their resistance to mildew, and an attempt is being made in the United Provinces to develop immune varieties.

The fungus is known on *Papaver Rhoeas*, *P. dubium*, *P. Argemone*, and *P. somniferum* in Europe and on *Argemone platyceras* in North



FIG. 143. *Peronospora arborescens*. 1, tip of conidiophore with conidia,  $\times 200$ ; 2, conidia germinating,  $\times 200$ ; 3, an oospore,  $\times 200$ .

America. Cases have been observed on *P. Rhæas* in Europe, in which the diseased plants gave flower-stalks whose buds failed to open, the stalks and buds, as well as the upper leaves, containing oospores but no conidia.

**Root rot** (*Rhizoctonia* sp.).—As already noted, some of the earlier descriptions of opium poppy blight suggest that this fungus was in part responsible, and it has recently been found to be very prevalent in poppy fields in some parts of Bihar and the United Provinces.

The symptoms resemble those already described under cowpea (p. 262). The disease appears chiefly in fields that are badly drained or impoverished, and may be found alone or in combination with the *Peronospora*. The plants begin to wither and dry up from the base. At the collar the stem is blackened and cracked, and the roots are sometimes also disorganised. The rot is usually a dry one, at least on the above-ground parts, and the leaves wither as a whole, not in spots as when attacked by *Peronospora*.

The tissues of the base of the stem and main roots are found to be infected with the mycelium of a *Rhizoctonia*, which agrees in its main characters with the small, black sclerotial form (p. 262), but has not yet been fully studied.

It is difficult to estimate the amount of damage due to this disease. It is worst, as a rule, under those conditions of cultivation which are least favourable to the poppy crop, whereas *Peronospora* attacks even flourishing crops.

Attention to the ordinary methods of good cultivation, especially drainage and manuring, would probably reduce the severity of the attack.

Besides the above diseases, the poppy sometimes suffers from a disease of a bacterial nature, which can be readily recognised. A soft rot commences at the apex of the stem and extends downwards, the whole plant breaking down ultimately into a soft, black, slimy mass.

### **TURMERIC** (*Curcuma longa* L.).

**Leaf spot** (*Taphrina maculans* Butl.).—This disease is common in many parts of India, perhaps everywhere that the crop is grown, having been recorded in Bengal, Bihar, the United Provinces, Bombay, and Madras.

It occurs also in Japan. In India it is found on several allied plants, including *C. Amada*, *C. angustifolia*, *Zingiber Casumunar*, *Z. Zerumbet*, and *Hedyochium*.

Though in severe cases every leaf is marked by hundreds of the brownish-yellow spots, the turmeric growers in the neighbourhood of Surat, where the most intense attack has been seen, do not consider it a

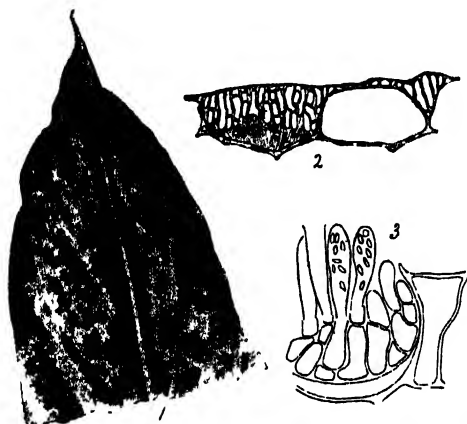


FIG. 144 Leaf spot of turmeric (*Taphrina maculans*) 1, part of leaf with spots,  $\times \frac{1}{4}$ ; 2, mycelium in the walls of epidermal cells; 3, asci and ascospores.

serious disease. This is probably because the attacked plants are not killed, the leaves retaining their vitality for a long time even when extensively affected. Since, however, the normal work of the leaf in assimilation must be much hampered by the presence of the fungus, it is probable that it is responsible for a reduction of the yield which may be much greater than the cultivators themselves suspect.

The spots appear on both surfaces of the leaf, often in great numbers, being generally more on the upper surface. They begin as a pale yellow discoloration, not sharply defined. This soon becomes dirty yellow, then deepens to the colour of old gold or sometimes nearly to a bay shade. The spots are small, usually from .1 to 2 mm. in diameter, and coalesce freely. They are not in any way limited by the veins and do not produce any local distortion of the leaf.

The hyphae of the parasite are embedded in the cuticle and in the cell walls of the epidermis and subjacent layer of cells. They never enter the cells, except to send large

branched or lobed haustoria into the cavities of the outer two layers of cells, and occasionally into the outermost cells of the spongy parenchyma (Fig. 15, 4).

Soon after infection, the first-formed hyphæ are found embedded in the cuticle which covers the epidermis, running chiefly in the furrows between adjacent epidermal cells, though some branches cross the tops of the cells. The hyphæ next form, by frequently branching, an accumulation of short-celled parenchymatous fungal tissue, chiefly in the angles between the cells, sufficient to raise the cuticle to a considerable extent. An extension from this often covers the entire epidermis of the spot, still wholly immersed in the cuticular layers. From the cuticular mycelium, flat bands of hyphæ pass downwards between the epidermal cells, the vertical walls of which are split to give them passage. These spread out in the inner wall of the epidermis, and also continue in the same manner down the vertical walls of the next layer of cells (hypodermis) to reach the walls in contact with the spongy parenchyma of the leaf. They do not penetrate further, except to send an occasional haustorium into the outer cells of the spongy parenchyma.

The accumulation of hyphæ in parenchymatous masses in the cuticle and vertical walls of the epidermis may be so great as almost to obliterate the cavity of these cells. Hence in old stages, the fungus appears, on cursory examination, to occupy the cell cavities, but the remains of the cavity bounded by the inner layer of the cell wall can usually be made out.

The formation of asci and spores takes place on both surfaces of the leaf, when the spots are fully developed and the central portion is occupied by an almost continuous layer of well-developed fungus elements in the cuticle and vertical walls of the epidermis. The outer cells of this layer grow out into cylindrical or club-shaped, thin-walled projections, which rupture the cuticle to reach the outer surface of the leaf and become asci. As a rule the ascogenous cells mature at different periods, so that the ripe asci are found in little groups, not forming a continuous layer covering the surface of the spot. All of the outer cells appear to be ascogenous. Below each ascus there is at least one basal cell, sometimes two or three in a row. The ascus itself is a protrusion of the inner wall of the ascogenous cell in the form of a thin-walled sac, rounded or flattened above, narrowed somewhat where it leaves the parent cell, and measuring, in its free part, 20 to 30 by 6.5 to 10  $\mu$ . It usually contains eight spores, but sometimes more have been found, formed doubtless by budding from the first-formed spores as is known to take place in allied species. The spores are hyaline, unicellular, ovoid or almost oblong, and 4 to 6.5 by 2 to 2.5  $\mu$  in diameter.

As no other method of propagation but these spores is known, spraying with Bordeaux mixture should be effective in checking spread, if applied as soon as the first spots appear. No experiments have, however, been carried out to test the economic value of spraying where this disease occurs.

### GINGER (*Zingiber officinale* Rosc.).

**Soft rot** (*Pythium gracile* Schenk\*).—This disease is the cause of considerable losses in parts of Gujarat (Bombay), near Rangpur (Bengal),

\*The name *Pythium gracile* Schenk has been retained, for the present, for this fungus, though it differs to some extent from the algal parasite studied by Schenk. The latter was not fully described, and there are several allied forms difficult to distinguish from it. The ginger parasite does not quite agree with any of these, and further investigation may show it to be a new species.



and in Malabar, well-known centres of the cultivation of what is ordinarily a most lucrative crop. It is not known to occur elsewhere.

The first outward indication of the disease is a general but slight paleness of the leaves of a shoot, followed by yellowing of the tip of the



FIG. 145. Soft rot of ginger (*Pythium gracile*) : healthy and diseased plants.

leaves. This yellowing gradually spreads down the leaf to the leaf sheath, often more rapidly along the margins. Behind it, the leaf progressively withers and dies, and ultimately hangs drooping from the

sheath. Then the whole shoot dries up. Meanwhile the base of the shoot between the rhizome and the surface of the soil turns a pale, translucent brown colour, and by the time that the leaves are well yellowed, it is very watery and soft, so that the whole shoot can easily be torn off from this point, though it rarely falls over spontaneously. The soft rot also extends beyond the collar into the rhizome, which is discoloured and gradually decomposes, forming a watery mass of putrifying tissue enclosed by the tough rind. In this mass the fibro-vascular strands lie isolated. The roots arising from the affected parts present in general the same symptoms. Once the disease becomes established, no new rhizomes are formed.

All the discoloured parts below ground, base of shoot, rhizome, and roots, contain the

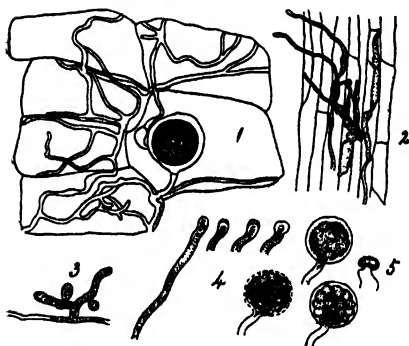


FIG. 146. *Pythium gracile*. 1, part of ginger rhizome showing mycelium with an oospore and antheridium,  $\times 330$ ; 2, mycelium in plant debris in water,  $\times 200$ ; 3, swollen lateral branch,  $\times 200$ ; 4, successive stages in zoospore formation,  $\times 200$ ; 5, a zoospore.

mycelium of the *Pythium* which sometimes ascends well above ground in the sheaths and stem. The hyphae penetrate the cells freely, as colourless, unseptate, much branched threads, up to  $4\mu$  in diameter and often with irregular swellings here and there. These hyphae can be traced beyond the limits of the parts which show obvious signs of disease, and always precede the rotting.

When a fragment of one of the lower scale-like sheaths (or of the rhizome), containing actively growing hyphae, is placed in water, some branches extend to the outside and form spores.

The apex of the branch swells up slightly, and after certain changes, which lead to its softening, opens suddenly by blowing out into a sort of bladder, resembling a soap bubble on the end of a tube. Into this bladder the contents of the hypha flow and become segmented into a number of zoospores, which escape by rupturing the bladder, and swim off by means of a couple of ear-like cilia. This spore formation can be readily induced artificially, and occurs naturally no doubt in wet weather, when the interstices of the upper soil are filled with water. Without a plentiful supply of water it cannot occur.

A second process of reproduction occurs within the tissues and results in the formation of oospores, whose germination has not been observed. The oospore results from the union of two cells, a large, spherical oogonium,  $27$  to  $33\mu$  in diameter and a smaller, beaked antheridium. After the contents of the latter pass into the former through the beak, the oospore develops as a separate cell, lying free within the oogonium. It is smooth, colourless, thick-walled, and  $23$  to  $27\mu$  in diameter. Antheridium and oogonium arise usually from different hyphae.

The fungus has been grown in pure artificial cultures at Pusa, and has been proved capable of attacking and rotting previously healthy rhizomes. The rotted tissues in the field are readily invaded by secondary saprophytes, fungi, insects, and eelworms, but at an early stage, and where healthy tissues have just been invaded, *Pythium graminicola* occurs alone.

There are probably two ways in which the crop may become infected. One, believed to be the commonest in Rangpur, by the use of diseased "seed"; the other, from oospores in the soil. If the seed contains any trace of living fungus, it is easy to understand how the newly-formed rhizomes and the shoot buds can get infected. This may happen by direct growth of mycelium into the new tissues, or by the germination of oospores still remaining in the scales and debris at the surface of the rhizome. The fungus is known to be a not uncommon soil-dweller and to be able to live as a saprophyte in soil, probably for considerable periods, so that underground spread no doubt goes on freely during the rains. How long the oospores can live in the soil is not known, but the period is probably considerable. They must reach the soil in large numbers with fragments of the lower sheaths and from diseased rhizomes left to rot in the ground as not worth lifting. Single plants, or even whole patches, are often thus left and must keep renewing the supply for a long time.

At Rangpur, the ginger "seed" (pieces of rhizome) is planted out in March and the disease is severe from the middle of August or September, when the plants are about 1½ foot high. It is said to appear first in July. After the end of September, spread is slight. It is evident, therefore, that its development is closely dependent on the monsoon. This is only what is to be expected, as the group to which the parasite belongs is semi-aquatic. In stiff soil, which holds water, the attack is always more severe than in sandy loam which drains well.

The damage done by soft rot is considerable in those places in which it has been investigated. Near Surat, in 1904, one village estimated its loss at 10,000 rupees. At Rangpur, in damp soil, there may be an annual loss of 10 to 15 per cent., while in dry soil the disease appears only in patches and the loss may be 5 or 6 per cent. Losses up to 80 per cent. may, however, occur in wet years. From November on, partially diseased rhizomes (being then nearly mature) are often dug up and sold for what they may fetch, presumably because they are not likely to give anything if kept until the usual harvest in January-February.

Treatment should be directed to preventing the supply of infective matter to the soil, and to improving the soil conditions so as to hinder the development of the parasite.

At harvest all rhizomes should be lifted. Diseased ones must be carefully collected and destroyed with as much of their roots as possible.

Ginger should not be grown again on the same land for several years. This is the usual practice in Rangpur, where the land is allowed to become overrun by thatching grass in between the ginger crops, which are from three to ten years apart, probably about five on an average.

Seed should be got from a place free from disease, and should be carefully hand-picked and all that seems to have any rot discarded.

Whenever disease occurs, the affected plants should be dug out whole, with the larger roots, and burnt. Pulling out is not satisfactory, as the rotted parts break off easily.

Good cultivation, and especially drainage, are of great importance, and also the choice of suitable soil. Light sandy loam and loam that is well drained are best. In heavier land, great care is necessary to avoid the accumulation of water in the upper layers, and the consequent increase in growth and reproduction of the fungus already referred to. At Rangpur, the cultivation and drainage seem to be alike very carefully attended to, and the soil selected for the ginger fields is usually light and open.

By the above measures the rot has been successfully kept in check at the Rangpur Experimental Farm. If regularly carried out everywhere, this destructive disease would probably lose most of its terrors.

*Pythium gracile* is a fairly common inhabitant of soil and fresh water, and attacks tobacco and probably castor and papaya, in addition to ginger, in India. In Europe, it is best known as an algal parasite.

### CHILLI (*Capsicum annum* L.).

**Die back.** (*Vermicularia Capsici* Syd.).—This is the most serious disease of chillies as yet observed in India, if we except an obscure disease, not yet studied, but believed to be similar to the mosaic disease of tobacco. It has caused considerable damage in Bihar of late years, and is prevalent also in Madras. Outside India, it has been recorded in the Philippine Islands.

The attack usually takes place in October or November in Bihar, when the crop is in flower. At this period individual flowers are found drooping and gradually drying up. Many of them fall off without further damage, but in favourable conditions for the growth of the parasite, extension occurs back through the flower stalk to the stem.

(Fig. 147, 2-3). Growth along the stem occurs rapidly, the bark turning at first brown and then white, in long, narrow strips. Lateral spread also occurs and the whole branch withers. When a fork is reached in the downward growth of the parasite, extension occurs both down the main

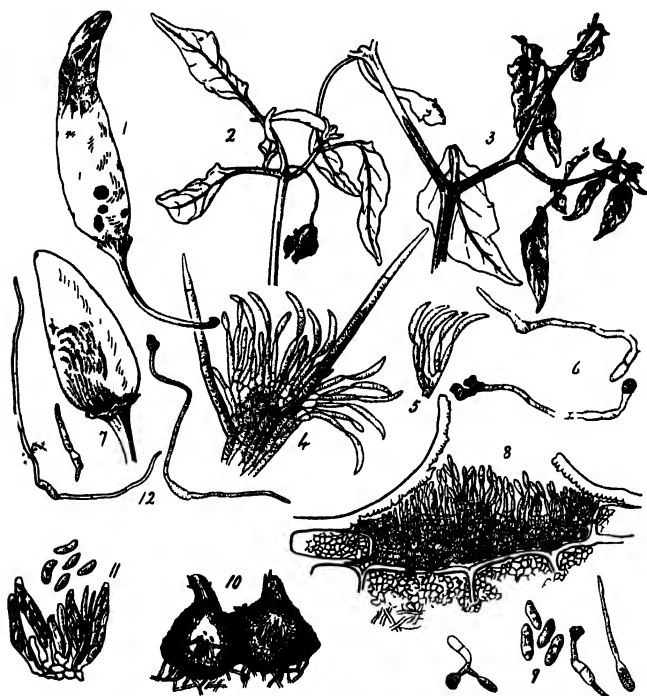


FIG 147. Die back (*Vermicularia Capsici*) and ripe rot (*Glomerella piperata*) of chilli: 1, fruit attacked by *Vermicularia Capsici*,  $\times \frac{1}{2}$ ; 2, early stage of die back, only a single flower affected,  $\times \frac{1}{2}$ ; 3, later stage showing extension from dead branch on right to main stem and branch on left,  $\times \frac{1}{2}$ ; 4-6, parts of sporodochia of *Vermicularia Capsici* showing conidia and setae,  $\times 355$ ; 6, germination of conidia of same, one with appressoria  $\times 315$ ; 7, fruit attacked by *Gloeosporium* stage of *Glomerella piperata*,  $\times \frac{1}{2}$ ; 8, acervulus of same,  $\times 245$ ; 9, conidia from same, and their germination,  $\times 365$ ; 10, perithecia of same,  $\times 75$ ; 11, asc and ascospores from same,  $\times 245$ ; 12, saccosporangia,  $\times 245$ .

stem and out into the healthy branch. In this manner the whole of the upper part of the plant may be killed, while the basal part is unaffected. Direct infection of main stems seems to be rare.

At a later period, further attacks occur on the fruit (Fig. 147, 1). These resemble those of the bean anthracnose described above (p. 256). The surface of the fruit is marked by roundish, dark depressions, which later dry and turn pale in the centre. These spread and run together, especially towards the tip of the fruit. The latter shrivels, and may dry up more or less completely.

The characters of the parasite closely resemble those of bean anthracnose, except that the woody tissues of the stem are more readily invaded, the hyphae being found in all the tissues. In the fruit, extension takes place through the walls, and the fungus reaches the seed just as in bean anthracnose.

The fructifications are produced copiously on the fruit and stem. From the internal hyphae, stromata are formed in the epidermis. These increase in size and rupture the cuticle, and then continue to grow so as to form a cushion-shaped or almost hemispherical stromatic tubercle on the surface. The surface cells of the stroma now grow out into two kinds of stalks. Some, scattered here and there on the stroma, grow into long, radiating, dark, rigid, sterile hairs (setae), while between them others grow into hyaline, short, closely crowded basidial stalks, on which conidia are borne (Fig. 147, 4-5).

The tubercular stromatic masses measure from 70 to 120 $\mu$  across, and are composed of violet-brown to deep brown cells, those at the base being elongated while nearer the surface they are polyhedral. The setae are dark-brown, lighter at the tip, with several septa, and up to 150 $\mu$  long. The conidia are borne singly on the top of the fertile stalks, which are unbranched and unseptate. They remain in a mass around the base of the setae after falling from the stalks, being surrounded by a mucilaginous substance which sticks them together when dry. The spore-mass is pinkish, but the conidia are hyaline individually. They are unseptate, curved, narrowed at the ends, and measure 17 to 28 by 3 to 4 $\mu$  in diameter.

On germination, a germ-tube is given from one or both ends, the spore often dividing first into two cells by a median septum (Fig. 147, 6). Appressoria, exactly similar to those found in the bean and other anthracnoses, are formed on the ends of the germ-tubes after a shorter or longer growth, and infection probably takes place usually by a hypha from one of these.

It will be seen that there is little real difference between this fungus and the *Colletotrichum* stage of species of *Glomerella*. In the latter, the spore-bearing acervuli are formed while the fungus is still immersed in the tissues of the host, so that it is one of the *Melanconiaceae*. In *Vermicularia*, the tubercles ("sporodochia") appear on the surface of the host before they bear spores, and they are often prominently convex, so that the genus belongs to the *Tuberculariaceae*. Whether such differences have any real significance is doubtful. No perfect stage has been found in *Vermicularia* as yet.

Dissemination is most likely to occur in moist weather, as the dry spores remain attached to one another. In Pusa, it is most noticeable that the disease develops with intensity at the end of the rains and while the heavy dews of the early cold weather still persist. Plants in shady localities, where dew is not deposited, often escape. The period of maximum damage is confined to a few weeks, and with the dry cold weather extension ceases.

Attempts made to control the disease by seed selection, as described under bean anthracnose, have failed. Spraying with Bordeaux mixture

has, however, given very promising results and is being fully tested in Bihar and Madras. It is possible that a single spraying at the end of the rains will carry the crop through the critical period of a few weeks when rapid spread goes on, and it has been found that even plants which have had the crown destroyed will put out new shoots from below and give a good crop, if these shoots can be preserved from infection by spraying.

Inoculations carried out at Pusa with pure cultures of the fungus have proved that it is capable of attacking a number of other plants, including tomato, brinjal, cowpea, and val (*Dolichos*). The fruit rots and die back of these crops (referred to under them) are at least in part caused by *Vermicularia Capsici*.

**Ripe rot** (*Glomerella piperata* (Stonem.) S. & v. S. = (?) *G. cingulata* (Stonem.) S. & v. S. = *Glæosporium piperatum* E. & E.).—The ripe rot is found usually (as the name indicates) on the ripe or ripening fruits of the chilli plant. It was first described in the United States, and recent workers in that country believe it to be identical with the common ripe rot of apples and many other plants, caused by *Glomerella cingulata*. If this is so (and there are no obvious differences observable so far as Indian material shows), then the fungus is of world-wide distribution and likely to occur wherever the chilli is grown.

The damage to the fruit in India is sometimes considerable. The injury resembles that caused by the last fungus when it attacks the fruit, except that the black or pinkish fructifications, which are just visible on the diseased areas, are often arranged in concentric bands (Fig. 147, 7). The attacked parts turn dark and become depressed or wrinkled, and ultimately the fruit shrivels and dries up. Attacks on the stem and leaves are rare.

The fungus resembles the last in many respects, but is not capable of extensive development in the stem tissues of the host.

The fructifications are formed under the cuticle. At first there is a large accumulation of stomatic tissue in the epidermal cells and extending down into the next layer. The cells are entirely destroyed and replaced by dark, short, polyhedral fungus cells. From the outer part of the stroma, just below the cuticle, a row of closely crowded basial stalks arises, and these, by their growth in length, raise the cuticle and ultimately rupture it. At the same time, each stalk bears a conidium at its tip, and probably new ones are formed when the first fall off (Fig. 147, 8). As in the last species, the spores remain in adherent masses united by mucilage, until thoroughly wetted.

The conidia are hyaline singly, pale pink in mass, unicellular, straight or slightly curved, with rounded ends, and of almost uniform diameter throughout. They measure 11 to 21 by 4 to 5.5 $\mu$ , the stalks on which they are borne being slightly longer than the spores. They germinate just as in *Vermicularia Capsici* (Fig. 147, 9).

The perfect stage is an Ascomycete of the genus *Glomerella*. The perithecia are in clusters, pear-shaped, membranaceous, dark-brown but lighter towards the tip, and

formed on or partly immersed in a loose stroma of light brown, interwoven hyphae (Fig. 147, 20). They open by a mouth at the end of a moderately long neck, which may be somewhat hairy. They measure about 250 to 300 by 100 to 200 $\mu$  in diameter, and contain numerous asci without paraphyses. They are formed both on the plant and in culture at Pusa.

The asci are clavate, sessile, 45 to 65 by 7.5 to 11 $\mu$  in diameter, and each with 8 spores arranged roughly in two rows (Fig. 147, 11). The ascospores are somewhat like the conidia, but often more curved and of less even breadth. They are unseptate, hyaline, with rounded ends, and 12 to 19 by 4.5 to 6.5 $\mu$  in diameter.

Inoculations with pure cultures of this fungus at Pusa have been successful in producing rot in fruits of tomato, brinjal, cowpea, val (Dolichos), citrus, plantain, and papaya, and on young sweet-pea plants; while in other countries it has been found to attack several of the common hosts of *Glomerella cingulata*.

As the characters of this fungus, and of the disease it causes, are closely similar to those of the anthracnose described below, remarks or treatment will be deferred to the next section.

**Anthracnose** (*Colletotrichum nigrum* E. & Halst.).—This disease has been found in Burma and probably occurs also in India. Like the last, it was first described in the United States and is probably of wide distribution. Indeed there is no certainty that it is not identical with ripe rot, to the extent of being caused by the same organism; for the only distinguishing feature between the two fungi is the presence or absence of hairs (setae) in the spore pustules. This distinction was, when the fungi were first described, considered to be of generic importance, but, as already mentioned under bean anthracnose, recent work has shown that it is not constant and that species of *Glomerella* may have conidial stages sometimes with and sometimes without setae. The perfect stage of *Colletotrichum nigrum* has not yet been found. If, however, it is obtained and agrees with that of the last fungus, there will be no sufficient reason to separate the two, and the most we can say is that the *Glomerella* of chillies has a conidial stage which is sometimes a *Gloeosporium* and sometimes a *Colletotrichum*.

The symptoms of chilli anthracnose are best marked on the fruit. At any time after the latter is about half grown, patches of decay appear, usually either at the tip or on one side. At first, while the fruit is still green, the spots are grey, changing gradually to brown. Sometimes there is a premature reddening of the fruit round the spot. The flesh below shrinks and the rind may be thrown up into concentric wrinkles. The fructifications of the fungus appear as black, erumpent points, arranged often concentrically. The whole flesh of the fruit may ultimately be



involved, becoming dry, shrivelled, and covered with the black spore-bodies. Such fruit may be shed or remain on the plant, but in any case is useless.

The fruit stalk and twigs are also sometimes attacked, often by direct spread from the fruit, but extension in the stem is more limited than in the back.

The microscopic characters of the fungus agree with those of *Glæosporium piperatum*, except that the acervuli are furnished with long, rigid, dark-brown bristles, up to 150 long by 4 $\mu$  broad.

Inoculation experiments, mostly in the United States, have shown that the anthracnose of peppers can be communicated to several of the common hosts of *Glomerella cingulata*, but it is not always clear whether *Colletotrichum nigrum* or *Glæosporium piperatum* was used. Both fungi have been considered recently by different workers to be identical with *Glomerella cingulata*.

The treatment, by spraying with Bordeaux mixture and other fungicides, does not seem to have given decisive results in the United States. There is, therefore, no guidance from outside as to whether spraying is likely to be profitable or not in India, and there is insufficient evidence as yet whether the spraying against *Vermicularia* will also reduce the amount of anthracnose. The latter disease does not seem to be as harmful as die back, and where the latter is absent it is doubtful if spraying is required.

Considerable differences in susceptibility to anthracnose have been noted in different varieties of chillies in other parts of the world. Thus, in British Guiana, the kinds known as "long white" and "long red" suffer greatly, while "red chilli" and "long bird" are unaffected. Amongst the great number of varieties grown in India, some are likely to show these differences, and could be introduced into cultivation where the disease is severe.

It is further recommended to collect and destroy all diseased fruit as soon as noticed, and to cut back the stems, if they are withering, as far as a healthy part.

#### PEPPER (*Piper nigrum* L.).

Stump rot (*Rosellinia bunodes* (B. & Br.) Sacc.).—Of the rather numerous diseases to which this valuable crop seems to be subject, this appears to be the only one whose cause has been ascertained, with any degree of accuracy.

It was first brought to notice in 1895, when specimens from Mysore were determined at Kew to have been attacked by a fungus allied to *Rosellinia necatrix*, the cause of root rot of the grape-vine in Europe. It was not until 1904, however, that the fructifications, by which only the species can be identified, were collected on the base of a tree killed along with the pepper in a spreading patch of disease.

The general characters of the group of diseases known as stump rot will be described in detail under tea (p. 435), as they have been most fully studied in that crop. It is sufficient to say that they almost invariably arise from the rotting stump of some jungle or shade tree, cut down without uprooting. Spread takes place chiefly in the soil, along the roots and also away from the latter, by strands of sterile fungus tissue,

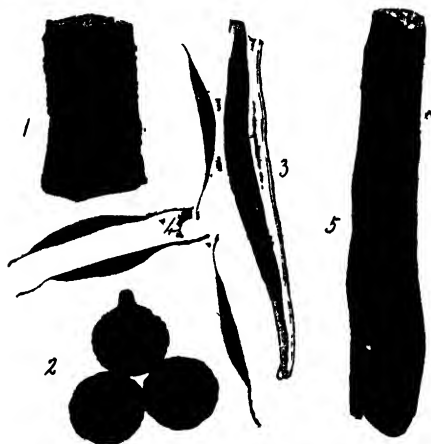


FIG. 148. Stump rot of pepper (*Rosellinia bunodes*): 1, perithecia at base of *Litsea angustifolia*,  $\times \frac{1}{2}$ ; 2, group of perithecia,  $\times 8$ ; 3, ascus and paraphysis from same,  $\times 200$ ; 4, ascospores,  $\times 230$ ; 5, mycelial strands on bark of *Holigarna longifolia* and blackening of wood under the bark,  $\times \frac{1}{2}$ .

often capable of extensive growth in length. Several species seem able to attack a number of different plants and to kill them by destroying the roots and outer tissues of the base of the stem.

In pepper, the patches of disease observed in Mysore are from 5 to 15 yards square, scattered through the plantation. The saplings and undergrowth of forest, and even large trees, within these patches, are also

often killed, amongst them being *Litsaea angustifolia*, *L. Wightiana*, *Schleichera trijuga*, *Holigarna longifolia*, and *Grevillea robusta*. Death is usually rather sudden; the leaves turn yellow and soon after fall, and the whole plant dries up.

The fungus attacks the roots and base of the stem below ground, appearing first as brown strands or continuous brown sheets, mixed with soil and often with grey, fluffy extensions of mycelium into the surrounding soil. From the superficial layers, hyphae pass into the deeper tissues in clusters and secondary strands, which flatten out into grey sheets under the bark. Other portions extend on the surface of the bark to above the soil level, where they give rise to small, purplish-black, velvety patches on the parts exposed to the air. On some hosts these patches form a continuous coating to the base of the stem for a foot or more above the soil, and even on dead twigs and leaves nearby, but it is not known if such extensive development occurs on pepper.

The superficial velvety growth consists of the conidial stage of the fungus, the conidiophores being grouped in short, black stalks, about  $\frac{1}{4}$  inch long, each hypha branching freely at the top and bearing minute, hyaline, unicellular spores. The ends of the branches are colourless and this, with the spores, gives a dusty grey appearance to the surface.

Later, the perithecia appear as round, black bodies, partly immersed in the velvety growth, but often ultimately free. They are up to about 1.6 mm. in diameter and with a slightly papillate mouth, the wall being ornamented with scale-like warts, regularly spaced and more or less concentrically arranged. The asci are up to about 300  $\mu$  long (sporeiferous part 270  $\mu$ ) by 12 to 15  $\mu$  broad, and contain 8 spores. These are long spindle-shaped, with unequal sides and the ends drawn out into a thread-like appendage, dark brown in colour, and up to 110  $\mu$  long (80 to 90  $\mu$  without the appendages) by 10 to 12  $\mu$  broad.

The part played by the spores in disseminating the disease is not known. It seems improbable that they can directly infect living plants, without first developing a mycelial growth in dead wood. Spores are only formed above the soil level, on a part of the fungus which is more or less saprophytic. The parasitic growth is sterile.

This fungus has recently been identified as a cause of the "black root" disease of limes in the West Indies and, together with an allied species, *R. Pepo* Pat., is also said to kill cacao, breadfruit, and several other plants. In Porto Rico, it apparently attacks coffee.

For treatment see under tea (p. 440).

### CORIANDER (*Coriandrum sativum* L.).

**Tumours** (*Protomyces macrosporus* Ung.).—This garden crop is much attacked in the neighbourhood of Pusa by the gall-forming fungus *Protomyces macrosporus*, which is known in Europe to infect many plants of the same order (Umbelliferae), including the carrot, parsnip, and caraway. It occurs also in north Africa and Australia.

The disease appears in the form of tumour-like swellings, which may be found on all herbaceous parts of the plant but occur most commonly in the flower stalks, leaf petioles, and green parts of the stem. The swellings are mostly elongated and may be up to  $\frac{1}{2}$  inch long by  $\frac{1}{8}$  inch broad. Their size varies as a rule according to the size of the infected

part, those on the stem being usually larger, for instance, than those on the flower stalks. On leaves, they are confined to the veins.



FIG. 149. Tumours on coriander (*Protoomyces macrosporus*): 1, shoots with tumours,  $\times 4$ ; 2, mycelium and chlamydospores in tissues of tumour,  $\times 215$ ; 3, germination of chlamydospore and first stage in spore-formation,  $\times 250$ ; 4, later stage of same with spores formed,  $\times 250$ ; 5, spores, some uniting in pairs,  $\times 250$ . (3-5 after de Bary).

The fungus is found only in the tumours. Here rather large, irregularly branched and closely septate hyphae occur between the cells. Their wall is thin and composed of cellulose. At an early stage, single cells here and there in the hyphae swell up to longish or oval, ultimately almost round, bodies, the chlamydospores (Fig. 149, 2). These become surrounded by a thick (up to  $5\mu$ ), colourless, 3-layered wall, and when mature are mostly 60 to 70 by 50 to 60  $\mu$  in diameter, but vary considerably. They are true resting spores, capable of germination after a rest of some months. They are formed in very large numbers between the cells, which may be so crushed and flattened as almost to disappear. The hyphae lose their protoplasm as the spores develop, and often can hardly be traced in old galls.

The germination of the chlamydospores, which only occurs if immersed in water, is peculiar. The outer wall ruptures and the thin, inner wall is pushed out into a sort of bladder, which remains at the mouth of the crack and contains the whole living contents of the spore. These segment in two stages into the final spores. The first stage begins by the collection of the protoplasm in a peripheral layer, leaving the centre empty except for cell sap, and ends by the formation of radially divided, uninuclear masses, arranged around the wall of the bladder in a single layer (Fig. 149, 3). Then, in the second stage, each of these masses (spore mother-cells) gives rise by segmentation to four spores, which, when fully formed, separate from one another and collect with the spores from the other mother-cells in a mass towards the centre of the bladder (Fig. 149, 4). The latter now bursts and the spores are thrown free into the water. Here they may, if brought on to a suitable host, give out a germ-tube which enters into the tissues and causes the development of a tumour; or if not in suitable conditions for resumption of the parasitic life, may continue to multiply vegetatively by budding in the manner of yeast cells (Fig. 149, 5), just as occurs in the smuts (see p. 179). Another point of similarity to the latter group lies in their faculty of uniting in pairs, like the paired fusion of the sporidia of *Tilletia* (p. 167).

The ejected spores are unevenly cylindrical or elliptical, 2 to 3 by  $1\mu$ , colourless, and non-septate. They have not been cultivated artificially beyond the yeast stage.

Infection seems to occur by the direct penetration of the germ-tube into the epidermal cells, not through stomata. All the hyphae below the epidermis are, however, strictly intercellular.

The effect on the plant is slight, except when the inflorescence is attacked, when the seed production may be diminished. There is evidence that the forms on different plants are specialised, some of those that have been tried being able to infect several hosts, others only one or two.

No treatment appears to have been tested anywhere, but the destruction by burning of all tumour-bearing parts should be effective in reducing the prevalence of the disease.

**Mildew** (*Erysiphe Polygoni* DC.).—Coriander is also attacked by this disease, which has been described already on peas and turnips (pp. 253, 300).

### **FENNEL** (*Faniculum vulgare* Mill.).

**Mildew** (*Oidiopsis taurica* (Lév.) Salm. var. *lanuginosa* Salm.).—The mildew of this crop is a variety of that already described on guar (p. 271), from which it differs only in certain microscopic characters of minor significance.

The chief of these are that the conidia are broad, oblong or cylindrical, tending to angularity at the corners, sometimes constricted in the middle, and 35 to 70 by 14 to 28 $\mu$  in diameter.

It should be checked by sulphur dusting.

### **FENUGREEK** (*Trigonella Fænum-græcum* L.).

**Mildew** (*Erysiphe Polygoni* DC. and *Oidiopsis taurica* (Lév.) Salm.).—These mildews have been already described on peas and turnips (pp. 253, 300), and on guar (p. 271). Both fungi attack fenugreek at Pusa, and one or other is found in many parts of India. They do little harm as a rule.

**Rust** (*Uromyces Trigonellæ* Pass.).—The uredo stage of this rust has been found at Poona, but nothing is known of the extent to which the crop is infected. It causes minute, brown spots on both surfaces of the leaves.

The sori break out from under the epidermis as small, powdery, cinnamon-coloured points. The spores are roundish, pale brown, and 20 to 23 $\mu$  in diameter. The wall is up to 3.3 $\mu$  thick, covered with scattered spines, and with several germ-pores.

The fungus agrees in its morphological characters with *Uromyces Anthyllis* a European species, but there is evidence that it is at least a specialised form or "physiological species" (see p. 60).

No treatment is known.

## CHAPTER X.

### FIBRE CROPS.

#### COTTON (*Gossypium* spp.).

**Mildew** (*Oidium* sp.).—This disease is rare in India, but has been met with occasionally in Bombay. It is the commonest leaf disease of the crop in parts of the West Indies, and the only observations on it come from that region.

Only the old leaves are attacked. Yellow or red, irregular patches, frequently bounded by the larger veins, appear on the leaf and ultimately spread over the whole surface. The under side is more or less covered by the white, shiny mycelium, which cannot be mistaken for any other fungus.

The fungus is only found in the conidial stage, and the species concerned is therefore unknown. It is said to have an internal mycelium and conidia borne singly on the conidiophores, characters which suggest that it may be identical with the mildew of guar (p. 271).

The damage caused is slight. The shedding of the older leaves is hastened but active leaves are usually untouched. Hence treatment is not likely to be required, but should it be, sulphur dusting would perhaps be effective.

<sup>1</sup> **Rust** (*Kuehneola desmium* (B. & Br.).—The cotton rust, usually known as *Uredo Gossypii*, from the stage ordinarily found on the leaves, is common at Pusa, and occurs in most parts of India, in Ceylon, Java, West Africa, the West Indies, North and South America, New Guinea and the Philippines. It does not seem to have been recorded in Egypt or Australia.

The fungus is confined to the green parts, chiefly the leaves, and has only been found in the uredo stage except in America, where the teleuto stage was discovered a few years ago. In India, the early stages of teleuto formation have been found but not mature spores.

The uredo sori are yellowish-brown, small ( $\frac{1}{15}$  to  $\frac{1}{4}$  mm.), pustular at first then more powdery; they are of two kinds, a primary on the upper surface of the leaf, rather deeply immersed in the tissues, and a secondary found underneath and more shallow.

The upper surface is often marked by small, purplish-brown spots which may run together into larger patches.

The sori develop under the epidermis, or more deeply immersed in the mesophyll, and are surrounded by a ring of colourless, clavate paraphyses (absent in the primary sori), which are often incurved so as to leave only a small opening. As a result of this, the sori bear a certain resemblance to an *Æcidium* and were, in fact, taken for such when first described in Ceylon.

The uredospores arise directly from the surface cells of a stromatic layer at the base of the sorus, and are without any definite stalk. They are oval or broad pear-shaped, light yellow in colour, with short but distinct spines on the wall, and measures 19 to 27 by 16 to 19 $\mu$  in diameter.

The teleuto is rare. The sori are deeply seated in the tissues of the under surface of the leaf, not readily distinguished from the uredo. They break through the epidermis

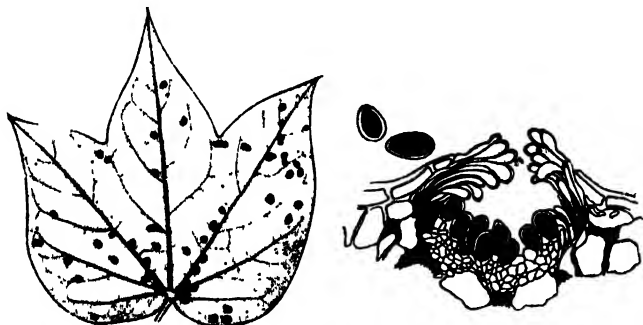


FIG. 150. Cotton rust (*Uromyces desmianus*): leaf with spots,  $\times$  5; section of uredo sorus, and uredospores,  $\times$  200.

at a late stage, and appear as somewhat powdery, light cinnamon-brown dots. The spores are cylindrical, and divided by 4 to 7 (usually 6) septa into a chain of cells which break away readily at the surface. The chains are cylindrical, laterally united below, and 80 to 110 $\mu$  long by 10 to 13 $\mu$  broad. The individual segments are irregularly oblong and 24 to 32 $\mu$  long. The wall is pale golden-brown, smooth, and thickened at the free end. A stalk cannot be distinguished.

The effect on the plant of this rust is to cause defoliation, but it has been observed that the fungus usually attacks weakly plants only. It is particularly found in India on introduced, unacclimatised exotics, and was also very prevalent, some years ago, on a collection of perennial and tree cottons from various parts of India and abroad, growing at Pusa. These did not thrive, and were attacked by a multitude of diseases and insect pests, which left the annual varieties almost untouched.

When severe attacks of this disease are observed in India, it may be suspected, as a rule, that the plants are not happy in their environment.



It is rarely found to any extent on well-grown field crops of indigenous varieties, or on exotics that have been able to adapt themselves to their new surroundings. Direct treatment, therefore, does not seem to be called for.

The fungus is not known on any other plant.

**Anthracnose** (*Glomerella Gossypii* (Southw.) Edg.).—This disease is rare in India, having only been found in lower Burma and once on seedlings at Pusa. It is exceedingly common and does some damage in the United States, and has been found also in the West Indies, Egypt, South and West Africa, Bulgaria, and Trans-Caucasia.

In its commonest form it is a boll disease, but serious injury has up to now only been reported from a few places; some cases are on record from the United States where the loss was from 50 to 75 per cent. of the crop, while losses of from 10 to 25 per cent. are more frequent. Besides the bolls, the leaves and stem are liable to attack, and seedlings have also sometimes been affected. The Burma cases seem to have been confined to the bolls.

The first symptom of attack on the bolls is the appearance of small reddish or red-brown spots, slightly depressed in the centre. As they enlarge, the sunken centre turns black, while the margin remains reddish. Several may occur on a boll and through their

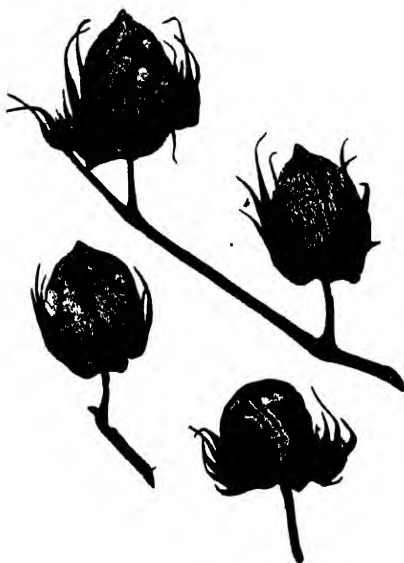


FIG. 151. Cotton anthracnose (*Glomerella Gossypii*): affected bolls. (After Atkinson).

union a large part of the surface may become involved. In damp weather, the centre of the spot soon becomes covered by salmon-coloured masses of moist spores, which dry into a crust. These may be

absent in dry weather. When the attack occurs before the bolls are fully grown, much deformity may result from the unequal growth of the healthy and diseased portions. The latter dry up prematurely, cease to grow, and crack so as to expose the lint. In such cases, the lint is usually ruined by the growth of the parasite or of other fungi into the interior of the boll.

When seedlings are attacked, the stem at or just below the soil level shows a reddish-brown discoloration, due to the death of the tissues at this point. Before the diseased spot has time to spread much round the stem, the seedling usually collapses. In this form of attack it is probable that spores of the fungus have remained adhering to the seed, and have been planted out with it.

On more mature plants, the stems are frequently attacked, especially at wounds and when the plants have been weakened from some other cause. The spots caused are rather like those on the bolls.

Leaves sometimes become infected, chiefly such as are sickly or wounded. The primary leaves of seedlings are especially readily attacked, often no doubt because of injuries received in freeing themselves from the seeds coats. On the leaves however, the harm done is slight as a rule.

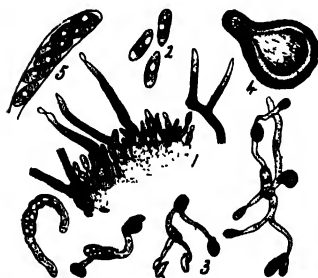


FIG. 152. *Glomerella Gossypii*: 1, part of an acervulus with conidia and setae two of which are fertile; to right, a branched seta,  $\times 245$ ; 2, conidia,  $\times 480$ ; 3, germination of same with appressoria and secondary conidia; 4, a perithecialium (diagrammatic),  $\times 100$ ; 5, acervulus with ascospores,  $\times 375$ . (3 after Atkinson, 4-5 after Edgerton).

The fungus is found in the cells of the blackened areas, causing their collapse and the gradual disappearance of the chlorophyll from green parts. Stromatic masses of fungus tissue of varying thickness soon collect below the epidermis, and give rise to the lower or *Colletotrichum* form of fructification of the parasite. The stromata produce basidia or spore-bearing stalks in large numbers on their outer side, and the epidermis ruptures or flakes off, exposing the spores to the air. The older stromata are dark-coloured, but the internal mycelium is colourless or at most slightly darkened. Basidia may also arise singly from the less deeply immersed hyphae, independently of the stromata, and reach the surface to produce spores; all sorts of intermediate stages between these single basidia and

true stromata are found.

The basidia are equal to or twice the length of the spores, broader below than above, usually unseptate but sometimes with one or two septa, and colourless,

They bear the spores singly at the tip, but after the first has been cut off, a second arises at the same point and so on until half-a-dozen or more have been formed.

The setæ are scarce or absent at first, then increase with the age of the stroma, and in old specimens are often densely clustered. They are dark brown below, colourless above, and arise from special large, thick-walled, dark cells at the surface of the stroma. They vary in length from 100 to 250 $\mu$ , are septate, straight or flexuous, rarely branched, and frequently bear spores at their tips, in this respect differing from those common to the genus, which are ordinarily sterile.

The spores are found both on the ordinary basidia and on the setæ. They are hyaline (salmon-pink in mass), unicellular, straight or less often curved, nearly oblong but with the ends rounded and the base usually narrowed, and very variable in size, from 11 to 20 by 4 to 9 $\mu$ . Those on the setæ are usually small and rather oval.

On germination, they generally develop a median septum and give out two or more germ-tubes, or sometimes form basidia immediately and produce spores quite similar to the original spore, from which they may be separated only by a short neck. The germ-tubes also soon form basidia and spores. In addition there is usually a more or less copious production of appressoria, which are especially numerous in unfavourable cultures. These bodies resemble those of the bean anthracnose, except that they are often composed of several cells. Their primary function seems to be to aid in infection, but they also probably serve as resting spores, though capable of giving out a new hypha immediately if conditions are suitable.

The perfect stage, an Ascomycete of the genus *Glomerella* to which most anthracnoses belong, has been found occasionally on diseased bolls in the United States and also grown in culture. The perithecia are immersed, dark, roundish or pear-shaped, sometimes with a prominent beak, and mostly about 140 by 115 $\mu$  in diameter. The asci are numerous, 55 to 70 $\mu$  long by 10 to 14 $\mu$  broad, and the spores elliptical, sometimes slightly curved, hyaline, not septate, and 12 to 20 by 5 to 8 $\mu$  in diameter. Numerous long, slender paraphyses fill all the cavity not occupied by the asci. This stage has not been found in India.

Artificial inoculations carried out in the United States and the West Indies show that the disease can be reproduced readily on young succulent parts, but on older leaves and stems a previous injury or lowered vitality is required. When the twigs are attacked, they turn black and their leaves change to various shades of yellow and green, then wither and dry up. Penetration frequently occurs through the scars left by fallen leaves. Infection of young seedlings may occur from the soil. Infection of the boll may occur directly from spores that reach its surface, or indirectly through the flower.

The vitality of the fungus is remarkable. Seeds from a diseased crop have had living fungus on them after more than a year. Dried cultures remain alive for many months. Diseased bolls and debris reach the soil from the previous crop, and the fungus probably has no difficulty in living over until the new seedlings are ready for infection. Spores entangled in the seed fluff must also often start the disease.

Hence the most important line of treatment is the destruction, by burning, of all diseased bolls, as soon as possible. If they cannot be

picked off as they are noticed, at least no débris should be left after harvest. For many reasons, not the least those connected with the depredations of insect enemies, the clean removal and burning of the whole of the plant at the end of harvest is of great value. Rotation is by no means universally practised in the great cotton tracts of the Berars and Khandesh, where cotton often follows cotton for several years. Anthracnose is fortunately unknown in these areas at present, but should it appear, the soil would probably become infected in a short time and require to be rested. Spraying is not hopeful against this disease.

The fungus is not known on any other plants.

**Grey mildew (*Ramularia areola* Atk.).**—This fungus is not common in most of the cotton tracts of India, having been found so far only on the east coast of Madras, at Pusa, and in Dohad (Bombay). It is known also in the United States, West Indies, and South Africa. It is confined to cotton.

It occurs chiefly on the older leaves, as the plants reach maturity,

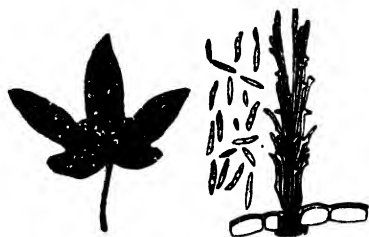


FIG. 153. Grey mildew of cotton (*Ramularia areola*): leaf with spots, under surface,  $\times 14$ ; conidiophores and conidia,  $\times 200$ .

in the form of irregularly angular, pale, translucent spots, 1 to 10 mm. (usually 3 or 4 mm.) in diameter, and with a definite margin formed by the veins of the leaf. As the spots grow older, the leaf tissues turn yellowish-brown, while a whitish, frosty growth appears, chiefly on the under surface but occasionally also above. This is the conidial stage of the fungus,

the only stage known at present.

The mycelium is confined to the spotted parts of the leaf and is entirely buried in the tissues except for the conidiophores, which emerge as clusters of parallel hyphae, measuring 50 to 150 by 4.5 to 7  $\mu$ , branching only at the base, and marked with prominent angular processes from the sides. They are colourless and divided by scattered transverse septa. Conidia are borne at the tips, and the lateral projections, which often all occur on one side, are caused by renewed growth of the conidiophore from below the insertion of the spores.

The conidia are formed at first in chains, later sometimes singly, and are colourless, irregularly oblong, with abruptly pointed (less often rounded or flattened) ends, continuous or divided by 1 to 3 transverse septa, and 10 to 35 by 4 to 5  $\mu$  in diameter. The chains are formed as in *Cladosporium*, the end spore being the youngest.

The disease occurs chiefly in low-lying, moist localities, and though the affected leaves are shed prematurely, little damage is caused as a rule.

Treatment is not required, so far as has been observed in India.

**Leaf spot** (*Mycosphaarella gossypina* (Cke.) Atk. = *Cercospora gossypina* (Cke.).—This minor affection of the leaves is found sporadically in many parts of India and, like the rust, occurs every year at Pusa, which is outside the true cotton area. It is known in the United States, West Indies, Egypt, and China.

It is said to attack only sickly leaves in the West Indies, while in the United States it is found usually on the older leaves but spreads over the whole plant in unfavourable weather, wet soil, or when the plant is not very vigorous. In China it is thought to cause great damage. The Indian experience agrees with that in the United States.

The disease is characterised by the appearance, at any stage in the growth of the plant, but chiefly when full grown, of spreading spots of roundish or irregular outline, visible on both surfaces of the leaf. The spots are, when single, up to about 4 mm. in diameter, at first yellowish-brown, then whitish in the centre from the withering of the tissues. The margin is formed of a distinct, dark brown or blackish rim which, in young spots, may be reddish at first. Adjacent spots may unite, causing large patches of the leaf to wither. In old spots, the centre often cracks or breaks away, leaving a perforation. Badly attacked leaves lose their green colour, turn pale, and finally wither and drop off.

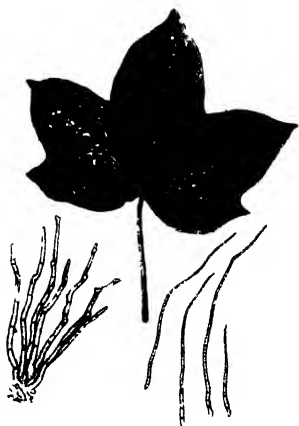


FIG. 154. Leaf spot of cotton (*Mycosphaarella gossypina*): leaf with spots,  $\times \frac{1}{2}$ ; conidiophores and conidia,  $\times 200$ .

The mycelium is strictly confined to the spots, the hyphae running between the cells. After growth for a time, small stomatic masses of dark hyphae collect near the surface, chiefly on the upper side of the leaf. From these, tufts of conidiophores emerge to the surface, usually through the stomata.

The conidiophores are numerous in each cluster, long (up to  $150\mu$ ), flexuous, dark brown, septate, and irregularly bent near the tip. The conidia are nearly colourless,

long, slender, curved bodies, narrow above and rounded below, divided by 5 to 7 cross septa and 70 to 180 by  $3\mu$ . Both spores and stalks vary greatly in size, according to the humidity and temperature during their development.

A perithecial stage of the Ascomycete genus, *Mycospharella*, has been found in the United States, where it is sometimes common on the older spots, and in China. The perithecia are ovate, black, partly immersed in the loaf, the mouth projecting on either surface, and are 60 to 70 by 65 to  $90\mu$  in diameter. The asci measure 40 to 45 by 8 to  $10\mu$  and contain 8 elliptical or broad fusoid, 1-septate spores. These are greenish-hyaline, the two cells often unequal, narrowed at the septum, and 15 to 18 by 3 to  $4\mu$ . This stage has not been discovered in India.

As to the treatment, the same remarks apply as in the case of cotton rust. The damage to the older leaves is almost negligible, and attacks on the young parts usually indicate want of vigour or an unfavourable season.

Other leaf discolorations frequently seen on cotton in India, especially on exotics, have not been traced to any definite fungus attacks. Some are caused by insects, others appear to be physiological, and in one case, seen in the Punjab, there was every reason to believe that the plants were poisoned by weak concentrations of alkali salts in shallow soil, overlying an impervious bed of lime nodules.

**Root rot (*Rhizoctonia* sp.).**—That this widely distributed parasite of cultivated plants in India is capable of attacking cotton has already been mentioned in the account under cowpea (p. 262) and groundnut (p. 323), where its general characters are described.

On cotton, *Rhizoctonia* causes chiefly a seedling disease. In serious cases, as in an attack observed in Dehra Dun in 1904, the mortality in the field may be considerable. At Cawnpore, in 1911, there were a number of cases in pot cultures of special varieties, but the local cotton was not attacked in the field. In Madras, in 1913, scattered cases were encountered in the fields throughout a considerable area in the north of the Presidency. The mature plant is also sometimes subject to attack in India and also in the United States, where, as well as in Egypt, the seedling disease is well known.

Infected seedlings usually show a soft, yellow patch on the lower portion of the stem, when the attack occurs while the seedlings are very young and the stem soft. In slightly older seedlings, when the stem is harder and more woody, it becomes blackened at the ground level. In bad cases, the rot is enough to cause tender seedlings to collapse, while older ones dry up and wither as if affected by wilt. Usually the attack

is of brief duration, and once the plants reach about 6 inches in height no further cases occur. Some seedlings succeed in shaking off the disease, and in this case a dry, brown spot persists on the stem.

The hyphae are found in the rotted area, at first in the cortex, later, in bad cases, extending in as far as the pith. Sclerotia are rare on the diseased seedlings. They were not found in the D hira Dun and Madras cases and only once from Cawnpore. Pure cultures of the fungus obtained from diseased seedlings, however, yield them readily, and they quite agree with those on other host plants.

Experiments show that different varieties of cotton differ in their susceptibility to the seedling disease. Thus, 90 per cent. were killed

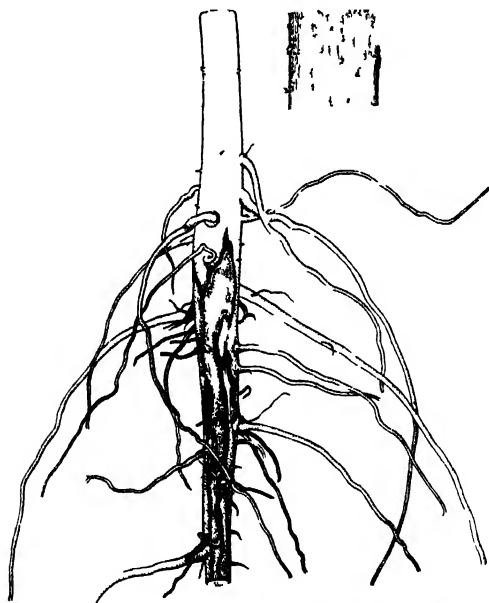


FIG. 155 Cotton root rot. Affected plant with bark removed from base of stem and roots,  $\times 1$ . Above, part of surface of wood showing sclerotia of *Rhizoctonia*,  $\times 2$ .

when the variety known as "khaki" cotton was infected, but only 20 to 30 per cent. when Cawnpore "desi" seed was used.

As already mentioned under those crops, the strains of the fungus from cowpea and groundnut can attack cotton but less vigorously than

its own strain, while that from jute is powerless to cause injury to the other three.

As regards the form of attack on the mature plant, it is not yet certain how far *Rhizoctonia* is responsible for the observed conditions, or how far it is secondary to some more serious adverse influence. In many parts of northern and western India, cotton suffers from a diseased condition known as "root rot." The base of the stem and the upper part of the tap root rots, the rot affecting at first chiefly the bast and external tissues, which break down into fibrous shreds. On peeling off the bark, the surface of the wood is found discoloured in a characteristic fashion, but the deeper parts of the wood remain unaltered for a considerable time. *Rhizoctonia* hyphæ and sclerotia, agreeing quite with those already described, occur in the deeper layer of the bast and on the surface of the wood.

It is noted that the disease occurs on the same patches year by year, and even after a rotation of 2 or 3 years. The patches vary in size from quite small to many yards across. Where the soil has been examined, it has usually been found that a layer of somewhat stiff clay, mixed with "kankar" (carbonate of lime) nodules, underlies the patch at from 1 to 4 feet.

The limited attempts that have been made, at Pusa, to reproduce the disease by inoculating large cotton plants with pure cultures of the fungus, have failed, and it is evident that if *Rhizoctonia* is the cause some special conditions regulate its attack. These may be connected in some way with the presence of the injurious alkali salts so commonly found in much of the area where the disease is prevalent.

The treatment of the disease caused by *Rhizoctonia* has already been discussed under cowpea (p. 262).

### **JUTE** (*Corchorus capsularis* L. and *C. olitorius* L.).

**Root rot** (*Rhizoctonia* sp.).—The only fungus disease of any importance hitherto found affecting this valuable crop is one caused by the *Rhizoctonia* with small, black sclerotia, described under cowpea (p. 262). It has appeared sporadically most years at Pusa, and has been found from time to time in the jute districts of north-eastern India. Few outbreaks have, however, been recorded as causing really appreciable damage, though our knowledge of these districts is too incomplete to enable us to be at all sure that such attacks may not often occur.



The plant may become infected at all stages of growth. Seedlings are very subject to attack at Pusa, the symptoms being those of a "damping off." Affected plants show first a slight, yellowish patch, just above the ground level; after a time this darkens and the tissues soften and collapse, the whole seedling falling to the ground. Young seedlings from one to two inches high are extremely susceptible to the disease.

Older plants are also attacked at or below the ground level. The infected area is easily recognised by its pronounced blackening; in this part the outer tissues are softened and partially disintegrated, but not enough to cause the stem to collapse; the sclerotia (Fig. 7, 3) are just visible to the naked eye. The leaves wither and the plant dies. Mature plants, on a superficial examination, look as if they had ripened before time; on pulling them out, however, the rot of the larger roots and base of the stem can at once be seen.

The characters of the parasite have already been described (p. 262). No morphological difference can be detected between the fungus on jute and on the other hosts previously mentioned. Nevertheless the jute parasite is unable to infect cotton, groundnut, or cowpea under normal circumstances; and the strains from these three hosts are not as readily parasitic on jute as its own strain, the cowpea strain, for instance, being only able to attack wounded seedlings of jute, but not the unwounded plant.

As the area under jute is expanding rapidly within some of the jute districts, the tendency to grow the crop frequently on the same land is on the increase. There is a danger that this may lead to an accumulation of the fungus in some soils. As already said, rotation and good cultivation are the only reliable measures to check damage by this pest. Recently, heavy manuring has been found on the Dacca Farm to reduce susceptibility to the disease in soil exhausted by overcropping with jute, and this line of enquiry is being followed up. Evidence has already been obtained that potash is the essential constituent in this case.

### SANN HEMP (*Crotalaria juncea* L.).

**Rust** (*Uromyces decoratus* Syd.).—This rust does a good deal of damage in the Godavari, and has also been found at Dehra Dun and Dacca. It is known in Ceylon on allied plants.

All parts of the plant above ground are liable to attack, the stem being often very severely affected, and the leaves, petioles, and fruit also involved.

Uredo sori are found on the under surface of the leaves, the upper surface being, in bad cases, marked by corresponding yellow spots, which may be absent in mild attacks. The sori are also numerous on the other green parts. They are scattered or crowded, yellowish-brown, powdery, often confluent, and surrounded by the remains of the ruptured

epidermis. The uredospores are round to ellipsoidal, dilute brown, with a spiny wall, and measure from 21 to 25 $\mu$  in diameter.

Teleuto sori are found in the same situations, at a later period. They are smaller than the uredo sori, black, and less powdery. The teleutospores are nearly round to oblong, with an evenly thickened wall, not specially thickened at the apex, but with a hyaline papilla at this point up to 3 $\mu$  in length. The spores are chestnut-brown singly, the wall provided with large warts, arranged in rows. They measure 20 to 28 by 14 to 20 $\mu$  in diameter, and have a stalk about as long as the spore, hyaline, slender, and easily detached from the spore.

As with most rusts, treatment is difficult. It is not known whether any varieties resist the disease.

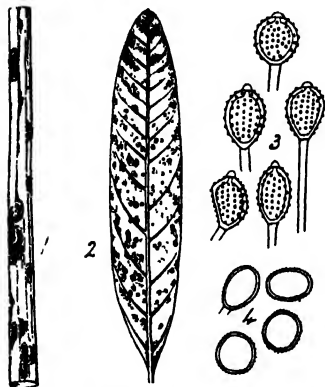


FIG. 156. Sann hemp rust (*Uromyces decortus*): 1, sori on stem,  $\times 2$ ; 2, sori on leaf,  $\times 2$ ; 3, teleutospores,  $\times 375$ ; 4, uredospores,  $\times 375$ .

### SISAL (*Agave* spp.).

**Anthraco**se (*Colletotrichum Agaves* Cav.).—This disease has been found from time to time

in various parts of India, chiefly on the true sisal (*Agave rigida* var. *sisalana*), but also on other species of *Agave* cultivated for fibre. It was first described in Italy, and is known in various parts of North America, in British Guiana, Guatemala, the West Indies, and British East Africa. In the important sisal cultivation of German East Africa, a disease with similar symptoms has been reported but is attributed to excessive heat.

Diseased plants are invariably affected by a curious blackening and withering of the leaves, which usually begins at the apex and extends down towards the base, but sometimes is restricted to a small, circular patch. The diseased portion of the leaf is shrunk, and thinner than the healthy and, as a result, the cuticle is thrown into ridges and furrows over the blackened area. In the more recently infected portions of the leaf, the colour is a light brown, which changes as the disease advances to deep black. In the final stages, small, erumpent nodules appear in concentric rings; but in the early stages there are no very obvious indications of parasitic attack.

Besides those cases associated with a fungus which has been experimentally proved to be capable of causing the symptoms described above,

others have been encountered where the most careful search has failed to disclose any parasite. It is not unlikely, therefore, that similar symptoms may be caused by conditions as yet unknown, and that the anthracnose is only one of a group of diseases closely resembling one another.

The hyphae of the fungus are found ramifying in all directions through the tissues of the diseased spots. After growth for a time within the tissues, they collect into thick masses under the cuticle of the leaf. From each of these masses, hyphae grow upwards in a brush like tuft and, finally, burst out through the ruptured cuticle as a mass of conidiophores, bearing spores. The leaf is sometimes capable of reacting to check the spread of the fungus within the tissues, by the formation of a layer of cork cells, across which the hyphae do not pass. In such cases isolated, more or less circular, circumscribed spots of disease are found. Two or more of these spots may become confluent.

The acervuli, after rupture, are covered in moist weather with an orange pink mass of extruded spores. The spore stalks (basidia) are closely crowded side by side, erect, sometimes branched, brownish at the base, colourless above, and variable in length but often 40 to 50 by 6 to 7  $\mu$ .

The spores are oblong, straight, with ends somewhat rounded, hyaline (pinkish in mass), and very variable in length those in India being mostly 13 to 20  $\mu$  while the Italian measurements are given as 22 to 26, and those in the United States as 16 to 31  $\mu$ . The breadth is from 4 to 6  $\mu$ . Amongst the basidia a limited number of sterile hairs (setae) is also found. These are from 65 to 170 by 5 to 6  $\mu$  (generally short in India), septate, yellowish to dark brown, lighter above, and with acute or blunt points.

Pure cultures of the fungus have been grown in the laboratory and successful inoculations obtained. At Pusa, it was found necessary to wound the leaf in order to secure infection. The first result is a blackening of the tissue immediately at the point infected. Spread now takes place, sometimes rapidly, the whole leaf turning yellow and the change in colour progressing from the seat of infection until the leaf ultimately becoming blackened, shrunk, dry, and with the cuticle thrown up into ridges; at other times the leaf resists invasion and only a small area is affected, leading to a black depression on both surfaces. The acervuli come out on the shrunk areas, usually in concentric circles.

In the field, infection appears to take place chiefly at the tip of the leaf.



FIG. 167. Anthracnose of sisal (*Collatotrichum Agave*): leaf with spot,  $\times \frac{1}{2}$ ; acervulus with conidia and setae (after Hedgcock).

No experiments in checking the disease have been recorded, but it is suggested that collecting and burning diseased leaves and spraying with Bordeaux mixture are methods which should prove efficacious in its control.

## CHAPTER XI.

### SUGARCANE (*Saccharum officinarum* L.).

**Smut** (*Ustilago Sacchari* Rabenh.).—This is the most easily recognised of the fungus diseases of sugarcane. It is known throughout the East, and also in the West Indies, Natal, and Italy. In India it is found chiefly in the north, but occurs practically wherever cane is cultivated. Only certain varieties are usually attacked, so that even in districts where it is not uncommon, individual growers may be unacquainted with it, because they do not grow susceptible varieties.

Affected plants are known by the production, from the growing apex, of a long, whip-like, dusty, black shoot, often several feet in length and much curved on itself. This shoot is an entirely abnormal growth, devoid of leaves, slender, and flexible. Most probably it represents a floral shoot, though often found in varieties that do not ordinarily flower in northern India and on parts of the plant, such as lower lateral shoots, where flowers are never borne. In its earlier stages, it is covered by a silvery-white, thin membrane, which soon ruptures, exposing a dense black dust, consisting of the spores of the fungus.

From the upper portion of the affected cane no secondary shoots arise, but from its lower part they are often fairly abundant, and may be all in their turn prolonged into spore-bearing organs. In such cases it is evident that the whole plant is infected. Sometimes, however, only a single lower shoot is thus transformed, the rest of the cane being normal; this, no doubt, indicates that localised infection of a part, only of the cane may occur.

The tissues of the cane below the abnormal shoot, and also the tissues of the latter, contain the mycelium of the fungus. The hyphae are found between the cells, and collect in dense masses towards the surface of the spore-bearing shoot. It is only here that spores are formed. The silvery sheath, covering the spores at first, consists of the raised epidermis (provided with hairs and stomata) and sometimes some of the subjacent layers of cells, and even a few fibro-vascular bundles. As the spores are formed in great quantity under the sheath, they rupture it, throwing it off in flakes and shreds. The deeper part of the shoot consists of large, thin-walled, parenchymatous cells, with numerous scattered fibro-vascular bundles.

The spores are spherical, smooth, light brown in colour, and 8 to 11 $\mu$  in diameter; mingled with them are little groups of colourless, thin-walled, sterile cells. They are disseminated by the wind, and germinate readily on falling into water or moist earth. On germination, a short promycelium is formed, usually straight and divided into two or three cells by transverse walls. Sporidia arise at the tip, and laterally near the septa. They are narrow, elongated, straight, single-celled bodies, joined to the promycelium by a short stalk but easily cast off. Under favourable conditions of nutrition, they can bud off similar sporidia in short chains from their ends. Sometimes, instead of bearing sporidia, the promycelium grows out into a branched hypha.

The exact details of the manner in which the cane is infected are

not fully known. It is probable that the disease can be transmitted through setts cut from diseased plants, containing living mycelium, as well as through spores. When totally infected plants are cut back to the ground level, the new "ratoon" shoots that appear from below ground, early produce spore-bearing shoots, sometimes when only a few inches high. Though it is possible that these shoots are the results of new infection by spores shed on the soil, it is more probable that they are caused by renewed growth of mycelium already present in the old stool. This is in agreement with experiments in Java and



FIG. 158. Sugarcane smut (*Ustilago Sacchari*): smutted shoot,  $\times 1$ ; spores and their germination,  $\times 250$ .

at Poona, which have shown that setts cut from infected stems give rise to smutted plants. Probably most of the early cases of smut, which are found at Pusa (where cane is usually planted in March) during

the hot weather and early rains, come from pre-existing mycelium in the setts used for planting.

The greatest proportion of cases of smut occurs, however, not early in the season, but when the canes are approaching their full growth and as they mature. These cases appear to be, for the most part, the result of spore infection. Artificial inoculations carried out in Java on the cut surfaces of the setts and also on the eyes, gave rise to diseased shoots in about a year. At Poona also, setts planted after being smeared with spores gave only a few cases after five months, but practically total infection was visible in eleven months. On the other hand, there appears to be good reason to believe that direct infection can be caused in the growing plant, at the point where, as we know from investigations of other diseases, the cane stem is least able to resist attack. This is in the vicinity of the joints or nodes, where the hard rind is interrupted by the shoot buds and rudimentary adventitious roots. If spores are placed inside the leaf sheath in contact with this part of the stem, a smutted lateral shoot may be obtained. Attempts to infect the cut ends of the setts have, in a few cases, given evidence that the fungus is able to enter and form a small mycelium in the pith, but our experience in India suggests that this is not so common as infection of the bud.

As a rule, the disease does not cause much damage, but the continued growth of susceptible varieties, once smut appears in them, is attended with much danger. No case of smut occurred in the cane crop at Pusa for the first three years of its history, and there was little for the next four years. A new lot of seed of Khari cane obtained in 1911, from a locality where smut was prevalent, led to a good many cases in the following year and the disease accumulated very rapidly, over 50 per cent. of this variety being smutted in 1913. The cases can be found from the first month after planting, and gradually increase in number up to the time of ripening. The varieties most subject to attack all belong to the class of thin canes so widely grown in many parts of India. Khari, Nanal, Saretha, Sonabili, and Katha all at times suffer severely, but no variety seems to be quite immune. Khari is perhaps the most susceptible cane cultivated in Bihar, and Saretha in the north-west. Of the thick canes, cases have been observed in most varieties, but no severe outbreak has as yet come to notice in this class.

No detailed experiments in the treatment of this smut have hitherto been carried out. It is safe, however, to recommend that all diseased plants should be cut out and burnt as soon as the attack is noticed, and

should on no account be used for seed. Until some reliable method of preventing infection is discovered, it is wiser not to grow the susceptible varieties named above once the disease begins to accumulate, but to replace them by those varieties known not to suffer severely, of which there is, fortunately, a plentiful supply in most parts of India. When the exact manner of infection is known, it may be possible to find some method of disinfection by fungicides, etc., such as are used in the cereal smuts, but preliminary experiments in Bombay indicate that copper sulphate is of little value and, besides, reduces the germination of the setts.

Besides sugarcane, the fungus is said to attack the wild species, *Saccharum spontaneum*, in Java and even to spread from the latter to the cultivated crop. In India, *Saccharum spontaneum* and *S. fuscum* are attacked by smuts which only differ from the above in the spore measurements. Whether they are the same or distinct species will only be ascertained when the cross inoculations are tried. The fungus on *S. fuscum* is eaten by cowherds in Assam.

**Rust** (*Puccinia Kuehni* (Kr.) Butl.).—The rust of sugarcane was first described as *Uromyces Kuehni* in Java, in 1890. Three years later, the same fungus was found to be the cause of cane rust in Australia. Later on, it was shown that the supposed *Uromyces* spores were only uredo-

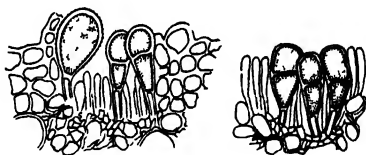


FIG. 159. *Puccinia Kuehni*: teleuto sori, that on left with a uredospore,  $\times 300$ .

spores. It occurs also in Japan, and probably elsewhere in the East, but does not seem to be known in the New World. In India, it is exceedingly common on a number of wild *Saccharums*, such as *S. arundinaceum*, *S. spontaneum*, *S. fuscum*, and *S. Narenga*, but has not yet been found on the cultivated sugarcane. It is difficult to believe, however, that Indian sugarcane is everywhere immune to this fungus and it may be expected to occur in some part or other of the Empire.

On sugarcane, only the uredo stage is known. This appears in the form of elongated, orange spots, which later turn more brownish. They are visible on both surfaces of the



leaf, on the under side being slightly raised above the leaf level. This is due to the formation of the sori, which, later on, rupture and expose the orange spore-powder. The uredospores are oval or pear-shaped, orange, and very variable in size, from 29 to 57·7 $\mu$  long by 18 to 34·5 $\mu$  broad, the average being about 48 by 27 $\mu$ . The wall is provided with short spines. At the margin of the sorus, paraphyses are found, which are club-shaped or cylindrical and of a brownish colour. On germination, the spores give a germ-tube which can infect the leaf by entering through a stoma. Before entry it swells up, and probably forms a sort of appressorium.

It has been observed that, in India, forms with the spore-wall equally thickened all round, and forms with a pronounced apical thickening, occur on some hosts, and in Japan both forms have been found in the same sorus. The latter form probably gave rise to the idea that the fungus was a *Uromyces*. A somewhat similar condition has been described above in the castor rust (p. 331).

The teluto stage has been found on *Saccharum spontaneum* in Burma and proves the fungus to be a *Puccinia*. The telutospores may occur in separate sori on the under surface of the leaf, the sori being small, elongated, and blackish; or they may occur intermixed in the uredo sori. They are accompanied by paraphyses similar to those of the uredo sori.

The telutospores are oblong to club-shaped, rounded or somewhat flattened above, narrowed below, not constricted or only slightly so at the septum, pale yellow in colour (? immature), and measure 25-40 by 10-18 $\mu$  in diameter. The spores are borne on very short stalks. Their germination has not been observed.

No suggestions for treatment can be given, but it is apparent that the varieties of sugarcane commonly grown are immune to the attacks of the forms of the parasite found on wild grasses in India. The case is evidently similar to the black rust of oats. Possibly even if the form capable of attacking sugarcane is found in India, certain varieties will prove resistant and can be grown.

**Ring spot** (*Leptosphaeria Sacchari* van Breda).—This is one of the leaf-spotting diseases of cane and, like all its class, does not kill the plant, but only reduces its vigour by injuring the leaf surface. It is not common on the thin canes usually grown in northern India, but is found everywhere that thick canes are cultivated. It was first described in Java, and occurs in most other cane-growing countries in the East, in Hawaii, the West Indies, and Australia. In Java it is found also on *Saccharum spontaneum*, a wild grass.

The first symptom is the appearance of small, discoloured, generally purple spots, visible on both surfaces of the leaf. These grow, drying up in the centre as they expand at the margins. The margin consists of a narrow, reddish-purple or brownish band, outside which there is sometimes a yellow areola, merging into the green of the leaf. The centre of the spot is dry, straw-coloured, and sharply marked off by the surrounding coloured ring. It is dotted with little black points arranged in rows, which are the fructifications of the parasite. The

spots are scattered over the whole or part of the leaf blade and, when full grown, may measure up to 15 mm in the direction of the length of the leaf by 5 mm in breadth. The ring is usually not regular, but lobed or broken by angular projections, and the union of neighbouring spots increases this irregularity. Ultimately, a considerable part of the leaf dies up in the neighbourhood of the spots, so that a feature of the later stages of the disease is the premature withering of the leaf.

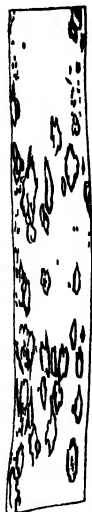


FIG 160. Ring spot of sugarcane (*Leptosphæria sacchari*) under surface of leaf with spots,  $\times 10$ , and part of upper surface of same, slightly magnified to show perithecia.

The leaf tissues at the spots are permeated by the fine, hyaline hyphae of the parasite which enter directly into the cells and kill them causing the contents to collect in a brown mass in the centre. At a later stage the mycelium extends into the withering portions of the leaf outside the rings. Perithecia are formed in large numbers chiefly within the rings but also to some extent, outside them. They occur as tiny black dots arranged in rows between the finer veins of the leaf and entirely buried in the leaf tissues except just at the tip. They are visible only on the upper surface of the leaf where they open by a fine mouth.

The perithecia are almost spherical about  $140\mu$  in diameter and contain numerous slender, elongated club-shaped ascus between which are fine paraphyses (fig 113 3). The ascus contains eight spores arranged in two or three rows. The spores are elongated 3-septate the two central cells especially the upper one thicker than those at the ends, straight rounded at the ends and about 20 to 24 by  $7\mu$  in diameter. They are colourless at first and light brown later.

A conidial stage has been described on the surface of the spots in connection with the same mycelium. It was at one time believed to be the chief agent in the spread of the disease the perithecia forming a resting stage to carry the fungus over to the next crop. More recent investigators, however, state that it is a separate fungus of merely accidental occurrence on the spots.

The damage caused is not considerable. A severe attack may do a good deal of injury to the leaves and, as noted in Hawaii, may give the cane a decided check. Such severe attacks are rare, but have been observed in Burma and occasionally elsewhere. Unfavourable conditions of soil and moisture appear to predispose the thick canes to it. Thin canes are never damaged.

No treatment has been tried, as it is unlikely that it would repay cost at present.

**Black rot** (*Sphaeronema adipozum* Butl.).—This is one of the commonest fungi found on cane in India, but as it is incapable of infecting

growing cane, and only attacks after ripening and usually after the cane is cut, the damage caused by it is slight. The same fungus is said to occur sometimes on diseased coconut wood in Ceylon. It has also been found on sugarcane cuttings received at Pusa from the United States, and one stage of it has perhaps been described, under the name *Catenularia echinata* Went, in Java, so that it may be widely distributed.

At the time of harvesting the crop, it is exceedingly common to find the broken ends of canes which have been injured by animals or the wind, covered with a greyish-black fungus growth, the cane pith itself being blackened at the point of injury. The same appearance may often be found on canes cut for seed, when kept for a few days. On splitting open the cane, the pith, deeper in than the blackened portion, is found dusky red in colour, the colour being usually in streaks. There is no obvious change in the exterior of the cane until a late stage, when the drying up of the pith causes shrinkage. At this period the pith is of a muddy brown colour, the redness disappearing gradually as the cane dries.

The discoloured pith is full of the hyphae of the fungus, which also grow freely in cavities in the pith caused by drying, and appear on any cut or broken surface

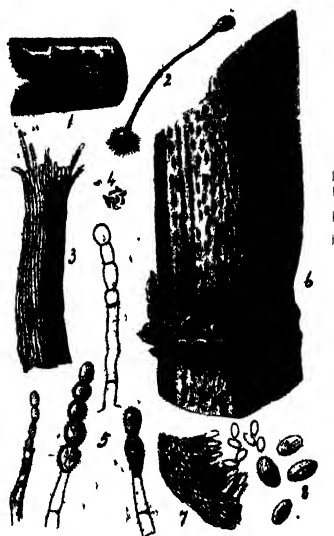


FIG. 161. Black rot (*Sphaeronema adiposum*) and dry rot (*Botryodiplodia Theobromae*) of sugarcane: 1, cut end of sett with pycnidia of *Sphaeronema*,  $\times 4$ ; 2, pycnidium of same,  $\times$  about 4; 3, neck of same,  $\times 280$ ; 4, pycnosporangia of same,  $\times 250$ ; 5, endoconidia of same,  $\times 250$ ; 6, cane stem with dry rot, nat. size; 7, part of pycnidium of same,  $\times 130$ ; 8, pycnosporangia of same,  $\times 250$ .

The hyphae are greyish-brown and penetrate the cells of cut or over-ripe cane readily. Conidia are found at the surface and in cavities in the cane. They are formed in the ends of short, special lateral branches, of which two or three may be united at the base, though more usually they are single. These branches are septate, usually forming a row of two or three cells, of which the end cell is the longest and bears the spores (Fig. 161, 5).

When spore-formation is about to begin, the tip of the hypha softens and swells up into a globular protrusion; at the same time, the portion of the protoplasm collected in this becomes cut off by a wall formed transversely in the hypha, just below the swollen part. The wall of the hypha then ruptures near the base of the swelling, and the nearly round mass is extruded as a spore. After the first spore is expelled, others are forced out from the open end of the hypha as fast as they can be produced, each remaining attached to the one behind to form a chain. Conidia thus formed within the conidiophore are termed "endoonidia."

The endoonidia are unicellular and extremely variable in size and shape, ranging from elongated, smooth, hyaline spores, to large, round, deep brown, spiny ones (Fig. 161, 5). Sometimes all the members of a chain are alike, especially in the case of those first formed, which are usually smooth and colourless, while those produced later are more often brown and spiny. Sometimes, however, the chain contains widely different members. The brown colour and spines of the larger spores only appear after they have emerged from the hypha. The wall in these spores is at first much thicker than in the smaller spores and is soft and jelly-like as they emerge. The inner layers then harden rapidly and turn brown, while the outer condenses in radial bands to form the spines. The chains may consist of 50 or more spores, and the latter vary in size from 9 to 25 by 4.5 to 18  $\mu$  in diameter, the smaller being hyaline and the larger coloured.

Later on, a second spore form appears. Large numbers of erect, rigid bristles, about a quarter of an inch in length by 50  $\mu$  broad, rise from the mycelium in places exposed to the air. At the base of each is a round, hairy receptacle, immersed partly in the mycelium. The whole forms a pycnidium, the round part containing the spores, while the neck is prolonged into a long, hollow bristle, opening by a mouth at the tip, around which there is often a short fringe of hyphae (Fig. 161, 1-3).

The spores are formed within the basal swelling, embedded in a foamy substance of a fatty nature, which is expelled with them in a translucent fatty drop at the tip of the bristle. The spores are in groups of from two to some dozens in this mass and are half-moon or crescent-shaped, hyaline, unicellular, and 6.5 by 3.5  $\mu$  in diameter (Fig. 161, 4).

Both types of spore germinate rapidly in water, giving rise to a septate mycelium which is similar in both cases, being hyaline at first and then greenish-brown. From each form, the other can be readily obtained in artificial cultures.

The fungus is not a strong parasite of cane. It is hardly ever found until the crop is ripe. At this stage it can attack the cane pith if introduced through a wound, but it is rarely found damaging the cane before it is cut. It is more destructive to the cut setts used for planting, but even here rarely kills the sett if germination is rapid. The worst injury seen was in the case of setts kept in earth pits for about a fortnight, before being used for planting. The fungus developed considerably in these, and a good many failed to germinate or died off soon after germination, white ants in most cases finishing the damaged sett before the new plant was established.

There is no treatment required, if proper care be taken to plant sound setts, and the conditions are favourable to early germination. If germination be delayed, the injury caused by white ants is so enormously greater than by any fungus disease, that it is not possible to say, until some effective method of checking these pests is found, whether any fungicidal

treatment would repay cost. Dipping the setts in Bordeaux mixture, as is done in some cane-growing countries, where other fungi injure the seed, has not been found to benefit the crop materially in India.

An allied fungus, *Thielaviopsis paradoxa* (de Sey.) v. Hoeh. is often found on pine-apples, causing a destructive rot of the fruit, and the same or a very similar form causes the "pine-apple" disease of cane setts in many parts of the world. The latter has been introduced into India on several occasions, but has so far failed to become established on cane in this country. Its pycnidial stage, showing that it also is a *Sphaeronema*, has been found in India, but not elsewhere.\* Another distinct but allied species is common on palms in India, and one or other of these is stated to be the cause of the stem bleeding disease of the coconut in Ceylon and the West Indies.

**Dry rot** (*Thyridaria tarda* Bancr. = *Botryodiplodia Theobromæ* Pat.). -The fungus which causes this disease is one of the commonest soil dwellers in cultivated land in India, and is an interesting example of the class of "weak" parasites, already several times referred to. Though undoubtedly capable of living as a saprophyte, it is more often found on dying or recently dead plants, than on old dead plant tissue. It is much easier, for instance, to obtain specimens by examining the roots of fairly recently cut back woody plants, when these roots are slowly dying in the soil, than by burying quite dead plant tissues. Apparently it requires for its optimum growth some slight degree of vitality to be left in the tissues it feeds on. Naturally, as a result of this, it is constantly being accused of causing the death of plants that have really been injured by some other agent. It unquestionably can kill healthy plants sometimes; much more frequently it merely hastens death, when the plant is already much weakened by other causes; sometimes it is a purely harmless saprophyte. Most observers have found it difficult or impossible to get it to attack healthy, unwounded plant tissues, and this has been our decided experience in India; we have also failed to get it to attack even the wounded tissues of plants such as indigo and orange when these were strong healthy examples, though it is commonly found completing the destruction of weakly individuals of these species; in other cases, such as rubber and sugarcane, it is an undoubted wound parasite; and a few cases have been encountered where it has attacked apparently quite strong, sound plants [see for instance under tea (p. 445) and rubber

---

\* Pycnidia are reported to have been obtained in cultures of the pine-apple rotting fungus in the United States, but have not been described.

p. 510)], so that it is unsafe to deny that it is sometimes a true parasite, not requiring wounds or weakness to facilitate its attack.

Though, in India, it is most often found attacking roots and the base of the stem, and we have therefore come to regard it primarily as a soil dweller, it is not uncommonly found also on stems and branches above ground; it is said, too, to appear readily on cut surfaces of pine-apple, potato, and other such material exposed to the air, in the vicinity of rubber estates in Malaya. Hence its spores must be freely present in the air. That it is not necessary for it to reach the soil to go through all its stages of development is also well established. It is as an inhabitant of the woody tissues of plants that it is best known, but it appears to be a matter of indifference whether these tissues belong to above-ground or under-ground parts; all that is necessary is that they should be accessible to the spores and belong to a suitable host plant.

The range of hosts and distribution are very wide. In India it has been found on sugarcane, tea, rubber, indigo, papaya, coconut, orange. In other countries on sugarcane, tea, rubber (*Hevea*, *Castilloa*, *Ficus*), coconut, cacao, coffee, camphor, tapioca, mango, papaya, *Albizzia moluccana*, *Erythrina lithosperma*, *Latania borbonica*; and the list of hosts is constantly being extended. It has only been recorded as injurious to sugarcane, tea, rubber, cacao, *Albizzia*, and *Erythrina*; in the other cases it is harmless or doubtful. It has been found almost throughout the tropics; in Ceylon, Malaya, Java, the Philippines, Samoa, South America, the West Indies, and West Africa.

On sugarcane it has been recorded from the West Indies, Java, and India. It does not appear to be capable of attacking healthy, unwounded cane, but has been proved to be able to attack through wounds, and it is not uncommonly found growing in canes suffering from red rot or other diseases. By itself it is not of much importance, but it probably adds to the damage caused by other agencies.

The disease resembles several other stem diseases of cane in causing little change in the external appearance until the pith is well permeated by the fungus. The tissues are turned blackish, the colour being largely due to the presence of the dark brown hyphæ in the cells. Later on, the affected canes become shrunk, and generally show longitudinal wrinkles from the falling in of the rind on the dried and diminished pith. The rind itself is raised into numbers of little, elongated blisters, which are arranged in rows, following each other closely in the row (Fig. 161, 6).

These blisters soon burst, and in moist weather extrude strings of spores from the mouths of the underlying pycnidia. In dry weather, the spores are not extruded in strings, but remain as a blackish crust overlying the ruptured areas.

The mycelium of the fungus is greyish-brown or dark brown in colour, only the very young hyphae being colourless. All the tissues are invaded, the hyphae passing freely through the cell walls from cell to cell. The main hyphae are rather straight and rigid, and often extend for considerable distances within the vessels, while shorter branches enter the parenchyma of the pith, and from these, fine, colourless threads ramify in every direction. The diameter is variable, averaging about  $5\mu$ .

When fructification begins, masses of brown hyphae collect to form dense stromata under the rind, usually in vertical lines. The pycnidia are formed in these stromata, sometimes singly, sometimes several embedded in a common mass. They are hollow receptacles, formed of an outer thick coat of brown cells and an inner one of colourless, thin-walled cells. From those of the latter which bound the cavity, short stalks project into the middle of the space. Some of these are fertile, bearing spores, while others, longer than the first, are sterile paraphyses (Fig. 161, 7).

The spores fall off as soon as ripe, and as fresh ones are constantly produced, they may eventually be forced out at the mouth of the pycnidium on the surface of the rind. When first formed they are colourless and undivided, but later on they become deep brown, and have one transverse septum (Fig. 161, 8). They are oval, not constricted at the septum, measuring 21 to 28 by 11 to  $14\mu$ , and borne on stalks about equal to the spores in length. The paraphyses are hyaline and up to about  $100\mu$  long.

On germination, which may occur while the spores are still in the colourless, undivided stage, a germ-tube grows out from one end, less often one from each end, and gives rise to a branched, septate mycelium in which the hyphae often unite by anastomosis. The young mycelium is at first white, but soon becomes coloured. Ripe pycnidia are formed in artificial culture on sugarcane in about two weeks from the germination of the spores.

Sugarcane has been artificially infected with this fungus in the West Indies. Only wound-infections have been described, pure cultures of the parasite having been inserted in small cavities under the rind. These produced a diseased area extending for about two inches from the inoculated point in four days, and pycnidia began to form in some cases within this period. Cane was also successfully inoculated in the same manner using cultures derived from diseased cacao, the internodes above and below the seat of inoculation becoming infected in eighteen days and pycnidia being plentiful. Further experiments proved that the fungus from cane attacks cacao equally readily.

It is stated that this fungus is the lower stage of an Ascomycete, named *Thyridaria tarda* Baner., which has been obtained in the stromata on cacao from West Africa, after about six months in a moist atmosphere, and has also been found on old rubber twigs (Heves) which had been infected with the *Botryodiplodia*. The proof is not conclusive.

Though not uncommon on sugarcane, it is probable that this parasite is not able to do much damage. It occurs usually in scattered cases, not in definite patches, and the cane is generally found to have been injured or weakened by some other cause. It has also been found on setts that had failed to germinate, but without any evidence that it was the cause of the failure. Should it ever become a dangerous enemy—a contingency always to be borne in mind in dealing with fungi of this sort—its

treatment will probably be in the direction of cutting out and burning all affected stems. As it does not occur on the leaves, and as there is no reason to believe that it can attack sound sugarcane through the roots this procedure will have a much greater chance of success than in such diseases as red rot.

The diseases of tea and rubber caused by this fungus are described below (pp. 445, 509).

**Collar rot** (*Hendersonina Sacchari* Butl.).—This is a disease which is sometimes confused with the much more frequent red rot. Though up to the present it has only been identified accurately in Jorhat, Samalkota, and Bangalore, it probably occurs in the Central Provinces and other central and southern parts of the country. It has not been found in northern India and is probably unable to survive the extreme changes of temperature of the more northern districts. It is not known outside India.

The symptoms outwardly resemble those of red rot; the top leaves wither back from the tip along the edges, the midrib remaining green later than the rest of the leaf. The larger leaves below the crown appear to suffer first, those at the apex remaining unaffected for some time. When the leaves have fully withered, the cane is found to be much lighter than normal. On splitting, the upper part is usually pithy and dry in the centre, or even with a central cavity along each internode, around which the pith is dry, white, and flaky. Lower down the pith may be still juicy, but has a curious translucent, watery appearance; still lower the central portion may be brown, while red streaks or patches may often be seen, especially at the nodes (Fig. 162). At the base, where the roots arise, the red colour predominates and is especially visible at the nodes. In old cases, the lower internodes also dry up and may develop a central cavity, surrounded by red or brown pith. The roots arising from the basal nodes are usually blackened and rotten, and the appearance suggests that the disease enters the base of the stem from the roots. Some cases were marked by a copious sprouting of the buds at the lower nodes, the shoots thus formed subsequently withering. It is uncertain whether this is a symptom of the disease or is due to a distinct condition, as it is not universally present.

The mycelium of the parasite is found in the reddened parts only, being absent from the translucent upper portion of diseased canes. Sometimes it collects chiefly at the nodes, the intervening internodes remaining almost free from hyphae until a late stage. The discoloured roots always contain a considerable quantity of mycelium. In the early stages, the hyphae run between the cells, being usually very fine, though thick ones





Fig 162 WILT (left) HELMINTHOSPORIOSE (middle) AND  
COLLAR ROT (right) OF SUGARCANE.



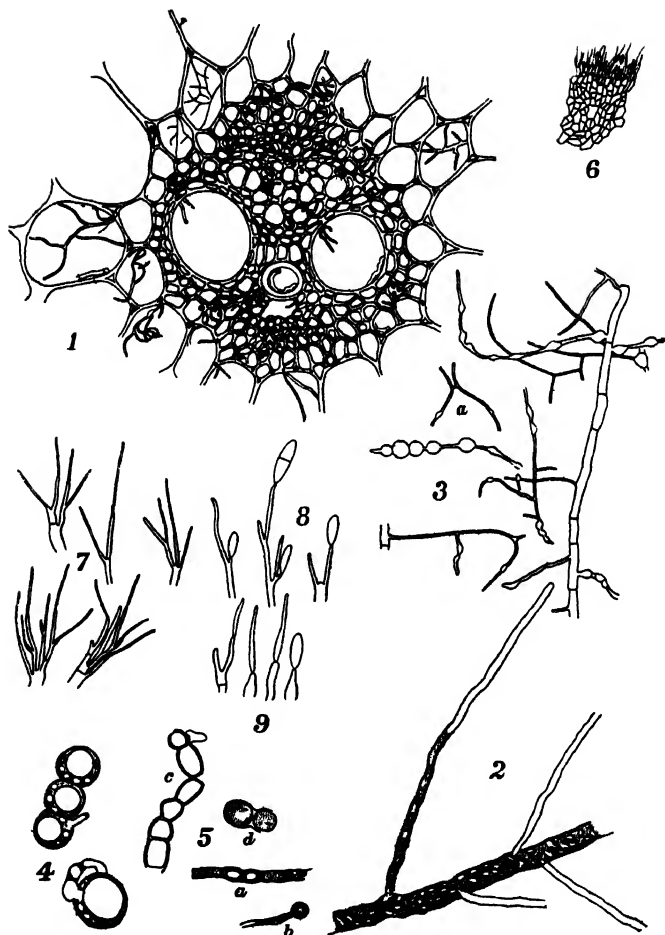


FIG. 163. *Hendersonina Sacchari*: 1, hyphae in pith cells,  $\times 145$ ; 2, thick hyphae with thinner branches,  $\times 260$ ; 3, finer hyphae with anastomosis *a* and chlamydospores,  $\times 260$ ; 4, chlamydospores, two germinating,  $\times 375$ ; 5, chlamydospores, *a* *b* early stages, *c* *d* ripe,  $\times 260$ ; 6, part of pycnidial wall with fertile layer; 7, sclerospores with branched stalks,  $\times 470$ ; 8, pycnosporos,  $\times 470$ ; 9, simpler types of stalk,  $\times 470$ .

sometimes are found. Later on, branches from the intercellular mycelium penetrate the walls to enter the cells. All the tissues are invaded, fibro-vascular bundles as well as parenchyma (Fig. 163, 1). In the cells, and especially in the vessels, very thick hyphae may occur, sometimes alone, sometimes intermingled with the fine filaments; both kinds belong to the same mycelium and can be traced into direct continuation with one another. At first septation is rather scanty (Fig. 163, 2), but in the older hyphae the septa lie very close together, the segments thus formed being often broader than they are long. No trace of spore formation has been found on the cane before death, though spores are formed on the old dead cane and also in artificial culture.

In culture, the mycelium is white or very pale yellow. The main branches are very thick (up to  $15\mu$  in diameter), at first hyaline and sparingly septate, then pale yellowish and closely septate. Branching is copious and often rectangular. From the older hyphae extremely fine, hyaline, thin-walled branches are given off (Fig. 163, 3). These are often irregularly swollen, or even nodular, and measure sometimes only  $1\mu$  in diameter. Intermediate stages, consisting of hyphae 6 to  $8\mu$  in diameter, freely septate when old and often irregularly swollen, are common. The thicker branches break up readily into oblamydo-spores (Fig. 163, 4-5), which may be terminal or intercalary, and are usually arranged in short chains. They consist of thick-walled cells filled with fatty matter, variable in size and shape, round to long elliptical, and up to  $33\mu$  in diameter, at first hyaline but later yellowish. They often separate from the mycelium when mature, and germinate in water readily.

Pyonidia are formed in cultures and have also been found on old dead cane. In



FIG. 164. *Hendersonina Sacchari*: 1, ripe sclerotium in transverse section; 2, pycnospores,  $\times 350$ ; 3, scolecospores,  $\times 350$ ; 4, intermediate types,  $\times 350$ ; 5, germination of pycnospores,  $350\times$ .

culture they appear in large, black, nodular stromata, about 1 to 2 mm. in diameter, composed of an outer portion of brown fibrous tissue, and an inner of dark brown parenchymatous cells, often many layers deep, within which spore-producing cavities (loculi) are hollowed out (Fig. 164, 1). On old canes, the stromata are found under the epidermis or somewhat deeper, at the internodes. They are smaller than in culture and without the fibrous covering. The base is broad, and extends all round as a rather narrow layer between the cells of the outer cane tissues. In the centre, the epidermis is raised up and ruptured by the roughly conical, deep portion of the fruit body, which is hollowed out into one or several loculi. Unilocular stromata are most frequent on the cane, multi-locular in cultures.

The spore-bearing loculi are deeply sunken in the stroma, very irregular in shape, and variable in number. They often communicate with one another by narrow passages, and sometimes several unite above into a common mouth, though each loculus may open independently. The mouths are formed late and are usually not prominent.

The cells bounding the cavity are lighter in colour than the rest of the stroma, and grow out into one or more hyaline sporophores ("basidia"), which are branched, irregular projections, each branch being pointed and ending in a single spore (Fig. 163, 7-9). Septa occur sparingly towards the base, the ultimate branches being always slender and unseptate. In some stromata, the basidia are mostly unbranched.

The spores are of two kinds, borne on exactly similar sporophores but apparently only one kind on any particular sporophore. One type, known as pycnospores, is brown, elongated, rounded at the ends, usually straight but sometimes curved, unseptate or with one or two transverse septa, and measuring 15 to 24 by 3.75 to 5  $\mu$  in diameter (Fig. 164, 2). Germination occurs by a germ-tube from one or both ends, the hyphae being at first unseptate (Fig. 164, 5).

The other spore form (Fig. 164, 3), the scolecospores, consists of hyaline, filamentous cells, usually without septa but with many oil drops, straight or irregularly curved, of uniform breadth, or tapering above, and 20 to 60 by 0.6 to 2  $\mu$  in diameter. They have not been germinated. An intermediate form of spore (Fig. 164, 4), is sometimes found, consisting of pale yellow, elongated, 1-septate cells, 18 to 30 by 2 to 3.75  $\mu$  in diameter.

All these spore forms may be found intermingled in the one loculus, or one or other may predominate over the others. Three-celled spores are never common.

Infection can take place both through wounds in the stem and also through the roots, the latter being probably the usual way in cane fields. Canes inoculated when more than half-grown in October, withered between January and March following. The parasite is much less virulent in Pusa than in Madras, the growth in wounded canes being slow and root inoculations without wounding having failed. It seems probable therefore that the disease is restricted in its distribution by climatic conditions, as so many of the fungus diseases of crops in India are.

The extent of the damage is not accurately known. Both at Samalkota and Jorhat it caused uneasiness on the Government Farms, but at Jorhat there may have been two diseases at work. There are many parts of India where cane suffers from a condition characterised by excessive sprouting of the lower buds and degeneration of the plant, sometimes to such an extent that no true canes are formed but only grass-like shoots. If it be shown that the present fungus is responsible for this, then it must be considered a cause of serious disease.

Probably the routine precautions urged in the next section in order to combat the spread of red rot should be effective in controlling this disease also.

**Red rot** (*Colletotrichum falcatum* Went).—The disease known as red rot in most English-speaking cane-growing countries, and as red smut

in Java, is undoubtedly the most serious to which sugarcane is subject in India. It is found throughout the country and is known in practically all other sugarcane-growing lands.

The first external symptom of the disease is that the upper leaves of a shoot, usually one approaching maturity, begin to lose colour and droop slightly. Then they wither at the tip and the withering progresses down the margins, leaving the centre green. It is usually not the young leaves at the tip of the shoot that are first affected, but the third or fourth from the top, these being the most prominent leaves of the cane. Later on, the whole crown withers and droops. At this stage there is no visible alteration in the cane stem and, except that withered and green shoots may occur in the same stool, the condition might be taken to be due to drought. Usually, especially in thick canes, when one cane of a stool shows the disease, the whole stool is ultimately, though often slowly, destroyed. Less often, and chiefly in thin canes, only a single shoot is affected. As will be seen below, the disease has a different origin in the two cases.

As the disease progresses, in severe attacks, large patches can be seen with every stool destroyed. By the end of the season little or nothing of the crop may be left undiseased. No other disease of cane seen in India causes such complete destruction, in bad cases, as this.

On splitting open a cane in the early stages of disease, a rather sour smell may be noticed, and the tissues are found to be reddened in one or a few internodes, usually towards the base. This discoloration, which is often present long before there are any external symptoms, appears first and is most intense in the vascular bundles, but ultimately extends to much of the cane pith. It is not evenly distributed, streaks or patches of darker colour separating lighter areas. Often definite red blotches, with a white centre transversely elongated, are found, and these are quite characteristic of the disease. In some outbreaks they may be absent from many individual canes. Several other fungi cause reddening of the pith and so do wounds in many cases, so that it is not easy to distinguish red rot in this stage by the unaided eye. Usually the reddening can be traced right down to the base of the stool, and in early stages, when only a single cane in the stool has begun to wither, the other outwardly sound canes may show red streaks rising from the base into the lower internodes. Occasionally, the reddening is confined to one or a few internodes in the centre of the cane, the upper and lower parts being free from disease. Spread to other canes in the stool does not occur in these

cases, which are due to aerial infection above ground and not to the usual type of infection from below. In the late stages, in both types of attack, the whole cane may be found infected.

As the disease progresses, the pith gradually dries up and changes to an earthy or brown colour. As a result, the rind falls in, in longitudinal depressions, and the cane becomes very light and easily bent or broken. Cavities develop in the pith, usually containing a more or less dense, cobweb-like growth of mycelium. When the cane dries up (and usually not until then), a careful search may reveal the spore acervuli on the surface of the rind. They appear as clusters of minute, black, velvety or hairy bodies, just above or below the nodes and also in the sunken areas found in advanced cases in the internodes, as a result of the shrinking of the dried pith.

The discoloured pith is full of the mycelium of the parasite, which can be found from the first at or below the ground level (in the usual type of disease), and gradually extends up through a great part of the cane. The reddening does not set in until a little time after the hyphae have extended into a previously healthy part, so that the mycelium can often be traced for a few inches beyond the discoloured area. The hyphae are chiefly found in the parenchymatous cells of the pith and in the spaces between these. They are slender, much branched, colourless, septate, and contain characteristic droplets of oil, which are sufficiently constant in appearance to enable the fungus to be identified from the mycelium in the pith with fair accuracy. The fibro-vascular bundles are less readily penetrated than the large sugar-containing cells of the pith, but hyphae can at times be found in the vessels and, more often, in the other elements of the bundle. The harder outer tissues of the cane are usually free from fungus until the pith has dried and conditions suitable for spore formation have occurred.



FIG. 165. Red rot of sugarcane (*Colletotrichum falcatum*): a, split cane showing pith markings; b, surface of cane with acervuli,  $\times 3$ .

The red colour of the pith is due chiefly to discoloration of the cell walls, often at some little distance from cells containing hyphae. The walls of the bundles are particularly prone to this change, and the position of the bundles is often marked by red streaks seen on splitting the cane, or dots on cutting it transversely. A reddish gum is sometimes present in the discoloured vessels, even when no hyphae can be detected in the immediate neighbourhood. Young parenchyma cells may have the cell contents changed to a similarly coloured, granular mass. When the pith cells are completely killed, the colour may fade in patches, and the white spots found in the reddened areas are due to large groups of dead cells, whose contents are replaced by air, whose walls have lost their red colour, and in which even the hyphae previously present have in great part disappeared.

Spores are often not easily found on diseased cane, and are rarely developed until

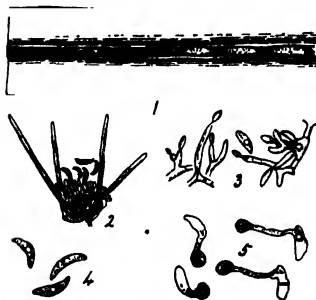


FIG. 166. Red rot of sugarcane (*Collatotrichum falcatum*): 1, attack on midrib,  $\times \frac{1}{2}$ ; 2, part of an acervulus,  $\times 130$ ; 3, formation of conidia in culture,  $\times 250$ ; 4, conidia,  $\times 250$ ; 5, germination of conidia with formation of appressoria,  $\times 250$ .

the cane has dried up. The mycelium collects into dark coloured, stromatic masses under the rind, especially near the nodes. The superficial cells of the stroma grow out into cylindrical processes, which, by their pressure, rupture the rind and appear at the surface (Fig. 166, 2). Some of these remain short and colourless, and form a crowded layer, bearing spores at the tip. The spore stalks are unseptate and measure about 20 by 8  $\mu$ , and the spores are colourless, unicellular, curved to a somewhat sickle shape, with narrowed ends, and usually a single oil drop in the middle (Fig. 166, 4). They measure 20 to 30 by 5 to 7  $\mu$  in diameter. Other cells of the stroma grow out into long, rigid bristles, dark below, lighter above, septate, and 100 to 200 by 4  $\mu$  in diameter. These "setae" may be scattered, but more

usually form a sort of fringe round the spore-bearing part of the acervulus.

Germination occurs readily in water or moist substances such as soil, one or two germ-tubes growing out from near the ends (Fig. 166, 5). The spore sometimes divides into two cells by a median septum, prior to germination. The germ-tube early shows the characteristic oil drops, becomes septate, and gives rise, under favourable conditions, to a branched mycelium. According to the conditions under which the spore germinates, this mycelium may grow for some time and reach a considerable volume, without forming anything but sterile hyphae, or, especially in dry or badly nourished cultures, may early give rise to chlamydospore-like bodies, now usually termed appressoria, since they have other functions than mere vegetative propagation. These may appear at any stage and in any position, from the moment of germination, when they may form actually in contact with the spore, to a late stage in the larger mycelial masses, such as are found in cavities in the cane or in artificial cultures. Usually they are terminal on a hypha, but they are also frequently found in its length. They are thick-walled, dark brown, unseptate cells, irregular in shape but often angular, with one or several large oil drops, and capable of being detached from the mycelium and germinating after a considerable time. They are produced in greater quantity in contact with the surface on which they are growing than elsewhere. In cases where artificial inoculations have been made with this fungus on cane, it has been observed that the penetrating hypha arises from an appressorium



and not directly from a spore (Fig. 17). It is, therefore, supposed that in addition to their function as resistant organs of propagation, they serve the double purpose of adhesion to the surface of the cane and accumulation of enzymic energy to secure penetration of the host. The old mycelial masses in cavities of the cane often bear large quantities of appressoria, and they are sometimes found in the cells and vessels of infected tissues. With the rotting of the cane they are set free into the soil, and can then resume activity.

Usually in artificial cultures, and also sometimes in the mycelial growths in cavities of the cane, true spores are formed at an early stage in the growth, differing only from those formed on the rind by being produced singly on short, lateral branches, and not from a stroma (Fig. 166, 3). Such spores reach the soil when the cane rots, and add to those shed from the rind. In the soil, when moist, the true spores do not last long, but spores exposed to the air and kept moderately dry germinate as freely after five months as when first formed. It is not known how long the appressoria live.

It is evident from what has gone before, that this fungus is not as well suited for air-borne dissemination, owing to the scanty and retarded production of spores exposed to the wind, as most of its class. There is, however, another part of the cane on which a quite similar fungus frequently occurs, and infection experiments leave little room for doubt that it is the same species. This is the leaves, where it is found both as a parasite of the midrib and as a saprophyte on old, withered leaf blades and sheaths, in both cases sporing freely (Fig. 166, 1). The midrib form has been found able to set up typical symptoms of red rot when inoculated into the cane pith, and very probably accounts for a large proportion of the *new* infections of cane above ground, as distinct from those which have arisen from the use of previously diseased setts or from the soil.

Infection of a healthy cane may take place in one or other of several ways. As cane is propagated by setts cut from a previous cane, we must distinguish between those cases where the disease is carried over in the sett, and those where it arises by infection by means of spores or mycelium from outside, through the soil, air, or irrigation water.

A previously sound cane can only be inoculated, so far as our experience goes, through wounds which expose the pith; through the scars left by tearing off unwithered leaves (as is sometimes done in "wrapping"); through the young buds ("eyes") or young shoots, but probably only when these are injured, as unfortunately is not uncommon especially at the time of planting; and, finally, through the adventitious roots, which allow the fungus to enter readily, even when quite sound and irrespective of position, those below ground being as freely penetrated as those not uncommonly found sprouting from the neighbourhood of the nodes. In the last case, the root "eye" above ground can admit the fungus, even before it has started to grow out into a root.

By far the commonest wound found penetrating the cane pith is that caused by the various species of boring insects which feed so freely on cane. In other countries, it is said that most cases of infection arise through this channel, but Indian experience indicates that, especially in northern India, borer holes are rarely infected. It is also uncommon in India to find infection occurring through leaf scars. Cases have been noted of infection through injured shoots, and this may perhaps occur also after the sett is planted. But probably new infections are most frequently caused by inoculation through the roots or root "eyes," in standing cane; and through these points, and also the cut ends of the setts, after the cane is planted.

Artificial inoculations allow the course of infection to be traced. Spores or mycelium may be used. If introduced into the cane through a hole in the stem, growth is rapid or slow according to the age and richness in sugar, and also according to the variety of the cane. Ripe or nearly ripe canes show rapid infection, while less advanced canes usually show only one or two joints affected after several weeks. Very young stalks may be killed fairly quickly, but the older ones often show little external signs of damage for several months, unless the inoculation has been made when they are nearly ripe. Cut setts are very rapidly permeated by the fungus; but if germination is prompt, the young shoot may grow away from the fungus, which remains in the basal part of the stool until late in the year. Often, however, the young shoot is killed, and red rot is responsible for a good deal of the failure to germinate and the dying back of young shoots, so common in certain kinds of cane in India.

The leaf scars are not liable to penetration once the corky layer, which cuts off the leaves prior to their complete withering, has formed. In the same way, the tough bud scales on young buds oppose the passage of the fungus for a time usually sufficient to allow the shoot to burst through the scales before it can be infected. But if the scales are bruised or torn, the hyphæ are able to pass into the bud and back into the main stem. Infection of the roots above ground is rapid, even when they are quite uninjured. The hyphæ enter directly into the epidermal cells, and through them pass to the deeper layers, reaching the stem in about a week. The active feeding roots in the soil are similarly penetrated through the unbroken epidermis, but whether the fungus can pass up into the stem from these roots is not known.

Leaves of sugarcane are sometimes attacked when quite sound, but the inoculation is more likely to succeed if the leaf surface be first slightly

wounded. When uninjured, penetration is chiefly into certain large, thin-walled cells known as "motor cells," which lie on each side of the midrib and between the veins. The thickened part of the epidermis is more resistant. If injured, penetration may occur into any part. As a rule, unless the injury is severe, the thick-walled sclerenchyma and the bundles long resist the passage of the hyphæ. In leaf inoculations, spore-bearing stromata may be formed at the surface in less than a week.

The methods of infection of the above-ground part of the cane stem described above are such as lead to single shoots becoming attacked, and the attack is usually confined for a time to one or two joints only, those above and below remaining free from disease. But, as already stated, by far the commonest type of red rot found in northern India is that in which the infection clearly arises from the base of the stool, and the whole clump ultimately becomes diseased. The epidemic outbreaks which have caused such destruction to thick varieties of cane have been almost without exception of this type. They arise from the use of setts cut from diseased canes, the parasite being carried over in the tissues of the setts.

When setts cut from diseased cane (and, therefore, usually showing the reddened pith at the cut ends) are planted, they germinate in most cases as well as if sound "seed" were used, unless the infection is severe and of long standing. But many of the young shoots begin to wither when they are a few weeks old, and the presence of actively growing *Colletotrichum* can be demonstrated in them. Many shoots, however, for a time grow away from the fungus, which remains dormant in the base of the cane. When the crop is about six months old and is beginning to contain an appreciable quantity of sugar, the parasite resumes activity, and from October on (in northern India) withered shoots are found in increasing numbers. By the end of the season many clumps, and even large fields, may be entirely destroyed. Periodic examination enables the fungus to be traced in the young shoot and, later on, rising from the underground parts into the large canes. Fields in which the "seed" is known to have come from a diseased crop will show much red rot, while adjacent fields planted with healthy "seed" (even of varieties known to suffer) may have little or none. This occurs to a marked extent in land not recently under sugarcane, but is also often seen where the crop has been repeated after three or four years. There is, therefore, no room to suppose that infection from the soil, and not from the setts, can be responsible for the disease in these epidemic outbreaks. Not alone has it been found that naturally infected setts can carry over the disease; but

perfectly healthy, growing canes have been artificially inoculated with pure cultures of the fungus obtained both from the leaves and the stem, and when subsequently cut up into setts and planted, have given rise to shoots infected with red rot.

The death of entire clumps in the manner just described, where the fungus is found rising from the base of the plant, is not in every case due to the use of diseased "seed." As already mentioned, the planted sett may become attacked if the parasite succeeds in reaching the cut ends, injured shoot "eyes," or the young adventitious roots. Artificial inoculations prove that even sound "seed" may give diseased clumps in this way, but, in northern India, the fungus does not seem often to reach the planted sett from outside, probably because spore-production is usually not copious and the parasite is unable to live long in the soil. There is some evidence, however, that where irrigation is practised, the water may carry infection from old canes and debris in the channels, and infected soil may be carried from trench to trench on the feet of the labourers.

We can see then that the normal course of the disease is somewhat as follows. Starting with a healthy variety of cane, there will be, in the first season, a limited amount of infection of above-ground parts by wind-borne spores, the fungus being found more or less in all cane-growing districts in India. There may also be a few cases of underground infection, especially if irrigation is practised. The following season, if no precautions be taken, each sett cut from a diseased cane will give a diseased clump, and there will be further new infections of the standing cane. This will continue with a progressively increasing accumulation of disease, until epidemic severity is reached, and it may be difficult to find enough "seed" for the following crop. Then, as a rule, new "seed" is obtained from some locality where the crop is healthy and the cycle recommences. Were it not that there are places where, for one reason or another, red rot is not common (as in the vicinity of Poona, for instance), the conditions in the whole of India would ere this have probably become like those in the Godavari Delta some years ago, where healthy "seed" was almost unobtainable and the crop was rapidly disappearing from the land.

The damage caused by red rot is more than the mere loss of a number of canes by withering. The disease may be present for a long time before withering sets in, and one sometimes comes across a field at or near maturity, where many of the canes show the characteristic discoloration

on splitting, though they are still unwithered and full of juice. It is well known to the cultivators that such canes yield a poor juice, which does not set readily on boiling. This is due to a remarkable power possessed by the fungus of inverting cane sugar and transforming it into glucose. The value of a cane largely depends for manufacturing purposes on the purity of its juice, that is on its richness in cane sugar as compared with glucose. The fungus forms a special enzyme, which converts some of the cane sugar into glucose and thereby spoils it from the sugar boiler's point of view. This enzyme is not only contained in the mycelium but is also secreted into the surrounding medium, and it seems highly probable that it is thus able to attack the sugar-containing juices of the cane, even at a distance from the actively growing mycelium.

In the control of this disease, the first requirement is to start with a healthy stock. In those districts where the local canes have become widely infected (as has happened in recent years in the Godavari Delta and in parts of Bombay and Bihar), new healthy canes must be brought in from outside. This has been done with remarkable success in the Godavari Delta, through the Government Sugar Station at Samalkota, where a large number of exotic and indigenous varieties have been tested, and also in Gujarat with varieties from Poona, and in Bihar with varieties from all parts of India and some from abroad. It is useless to start with a variety from stock known to have been seriously infected, as it appears to be exceedingly difficult to rid such a stock of disease by any practicable method of selection. It is well, therefore, for each important sugar-growing tract to be in a position to obtain healthy varieties when required, either by means of private nurseries where new kinds can be grown and old ones kept rigorously healthy, or by the now numerous Government Farms.

Systematic and thorough selection of the setts used for planting out should then be done each year, or the new varieties will not maintain their freedom from disease for long. The most convenient practice is to accept any red discoloration showing at the cut ends of the setts as a warning of disease. Other diseases and wounds may cause reddening of the pith, but there are advantages in rejecting these also, and it is well to be on the safe side. The preparation of the setts should be entrusted to experienced men, as it is important for several reasons that the ends should be clean cut and not shattered; they are easier to examine, and are less liable to admit parasitic or rotting organisms, if the part left between the end joints and the cut surface be uninjured. The cut

setts should then be collected into bundles of ten or fifteen setts (this is done at Pusa by boys) and submitted for examination by rapidly showing the cut ends to a responsible person, who should remove any with reddening of the pith. Even the cutters themselves soon get into the way of rejecting the most obviously reddened canes. Further, in harvesting the seed cane, much subsequent trouble may be saved by leaving out all clearly diseased clumps or patches, which can afterwards be destroyed or fed to cattle. They should on no account be left standing or ploughed in, as this only increases the spore production, and the fungus can remain alive in old canes for a long time.

Though reliance must be placed on the absence of obvious reddening at the cut ends in selecting setts for planting under field conditions, it has been found that, where there has been severe disease in the cane used for "seed," a certain number of diseased setts escape this precaution, owing to the presence of the fungus beyond the limits of reddening in recently infected canes. This is why it is necessary to start with good "seed," and not to use a severely infected field as "seed," even where expert selection is practised. This is also why it has not been found possible to eliminate the disease entirely, even in the form which is carried over from the previous crop and is not merely the result of new infection of the growing cane through the air. It must not be supposed that red rot can be got rid of in its entirety by any method of selection; the most that can be claimed is that it can be kept within reasonable limits in normal years and with good cultivation.

Of lesser importance, but still worth doing in most cases, is the regular removal of all withering clumps during the growing and ripening season. Such clumps, if left, dry up and produce spores, sometimes in considerable quantity. In some places (as in parts of Madras) it appears that the danger of aerial infection from such withering canes is considerable, though in northern India it has not been found frequent. There is also danger that the soil and, especially, the irrigation water may become infected.

Where the disease is prevalent, the practice of ratooning is, as will be evident from all that has gone before, exceedingly dangerous. Not only is time given for the disease already present, perhaps only in a mild form, in the stool, to penetrate all parts of the new growth, but the exposed cut surfaces allow a ready entry to the mycelium from spores formed on rotten canes and cane debris left after harvest. Ratooning

is not very common in India, but several cases have been seen where the second growth of a diseased crop has been almost or quite lost from red rot.

The value of a long rotation in cane cultivation is well established, especially in the heavy paddy soils of the Madras Delta and similar localities. A rotation of eight or nine years has been found beneficial in these cases. In the lighter soils of Bihar, cane one year in five, when irrigated, and even less if irrigation is not practised, has given good results. It is very doubtful, however, if this is connected with the persistence of the fungus in the soil, and not rather with the general vigour and resistance to attack of the cane.

In spite of these precautions, serious attacks of red rot, from circumstances not ordinarily under control, may occur. A few such cases have been observed. The attack of certain insect pests (cane fly, leaf hopper, and the like) seems so to weaken the vitality of the cane as to cause it to fall a ready prey to infection from without. Excessive flooding or the growth of the cane in water-logged soil may have the same effect. The action of defective soil conditions or bad cultivation in predisposing to disease has been mentioned in the general part (see p. 127) and need not be further referred to here.

It early becomes obvious to any observer of sugarcane diseases that there are immense differences in the susceptibility of canes to attack. The thin canes, so commonly grown in India, are, on the whole, much less liable to red rot than the thick. Their resistance seems to be inherent, whereas the temporary resistance of certain classes of thick canes, which has been observed from time to time, appears to break down readily under unfavourable conditions of the environment (see p. 127). It is only within the past few years that an opportunity has arisen of endeavouring to breed truly resistant canes of good quality in India, and though we look with confident hope to the achievement of success in this most important direction, it is as yet too soon for definite results to have been obtained.

In India, red rot is in many places the greatest obstacle to successful cane cultivation. In Madras, Bombay, and Bihar, the area under thick cane has, in certain districts, periodically shrunk as a result of an accumulation of this disease, to expand again only when the diseased cane has been replaced from outside. Red rot has often been the limiting factor in the successful cultivation of heavy-yielding canes, such as would enable

India to hold its own against other sugar-producing countries, like Java and Mauritius.

**Wilt** (*Cephalosporium Sacchari* Butl.).—This disease is sometimes confused with the last, as it produces distinct reddening of the cane pith. With practice, however, it is easy enough to recognise the differences between the two. It has only been definitely recorded in India, where it is found in most parts of the country, and in the West Indies. Some canes imported from the United States a few years ago were found to be infected by it, so that it is presumably present in that country also.

As in red rot, the earlier symptoms are elusive. Little is usually noticed until the cane is half grown, when stunted clumps, or even patches of varying size, may be observed. These gradually wither, and from this on, until harvest, withering of single canes or whole stools continues to occur. The leaves dry up, as if suffering from drought, followed by the stems, which become light and hollow. On splitting the cane when it first shows the disease, the pith is found to have a diffuse purple or dirty-red colour (Fig. 162), which is often streaky, but never in lighter and darker patches, and never with the transversely elongated white areas, characteristic of red rot; the colour is also not so bright as in that disease, and early becomes muddy and different from any other cane discoloration hitherto observed. In old cases it is an earthy brown. The pith dries up more rapidly than when attacked by *Colletotrichum*, and becomes hollow, the cavity often containing a fluffy, grey growth of mould.

The mycelium is found throughout the reddened portion of the stem, which may be only a few joints or the whole length. The hyphæ penetrate all the cells readily, bundles as well as parenchyma (Fig. 167, 2-3). They are usually fine, not exceeding  $3\mu$  in diameter, and, even when old, remain hyaline. Occasionally conidia are found on short lateral branches in the vessels, resembling closely the microconidia found in a similar situation in the *Fusarium* wilts of such crops as cotton and pigeon pea (Fig. 167, 5).

The greyish-white hyphal growth usually found in the hollows which always form in the internodes at a late stage, resembles that in the pith, though the hyphæ may be up to  $5\mu$  in diameter. They usually bear great numbers of conidia. This free mycelium is composed of even hyphæ (not abruptly swollen), without the oil drops so characteristic of *Colletotrichum falcatum*, without chlamydospores or appressoria, richly branched, and sometimes laterally united into narrow strands (Fig. 167, 6). Septa are few at first.

The conidia are hyaline, and borne on short, simple or branched, lateral hyphæ and also terminally on the end branches of the mycelium (Fig. 167, 6-10). They are 4 to 12 (usually 5 to 8) by 2 to  $3\mu$  in diameter when formed, but increase in size prior to germination. Their shape varies from short oval to long elliptical, and occasionally they are curved or with one side flattened. They are almost always unicellular when formed, but some become provided with 1 to 3 septa before germination (Fig. 167, 11).



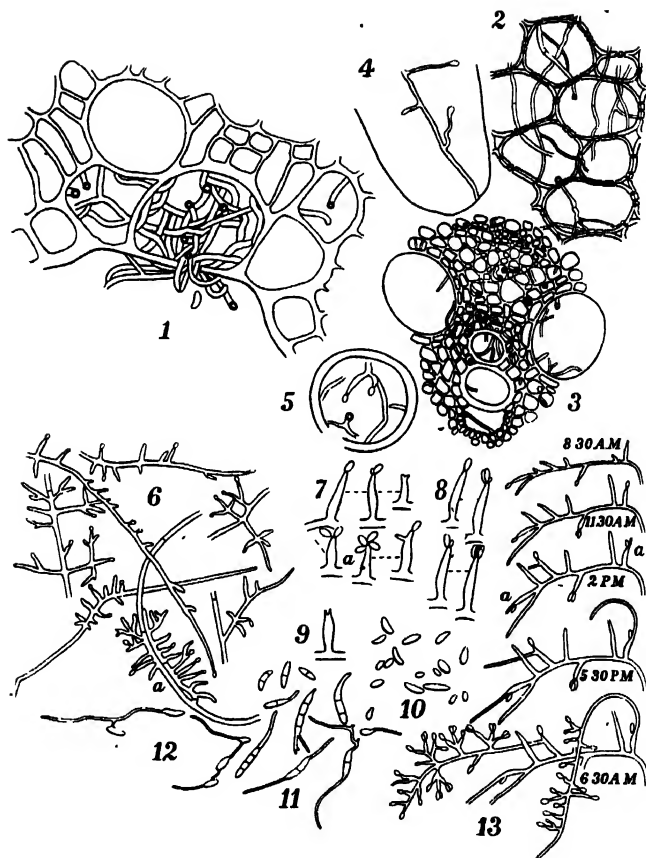


FIG. 167. Sugarcane wilt (*Cephalosporium Sacchari*): 1, fungus emerging from motor cells of leaf,  $\times 480$ ; 2, hyphae in pith,  $\times 145$ ; 3, hyphae in a bundle,  $\times 145$ ; 4, indications of conidium-formation in a pith cell,  $\times 240$ ; 5, conidium-formation in a vessel,  $\times 240$ ; 6, sporiferous mycelium, at a commencement of a coremlal strand,  $\times 240$ ; 7, conidiophores and conidia; 8, same, successive stages,  $\times 480$ ; 9, conidiophore showing hollow left after spore has fallen,  $\times 480$ ; 10, conidia,  $\times 480$ ; 11, germination,  $\times 240$ ; 12, anastomoses of germ-tubes,  $\times 240$ ; 13, growth of a sporiferous hypha, at a the conidiophore has continued growth after forming a spore.

The conidiophores are usually of definite shape, slightly swollen in the middle, with a rather blunt end, and often irregularly branched or once whorled or forked. They may be scattered or arise in bunches near together. If kept very moist, the tip may grow on into a hypha even after producing a spore (Fig. 167, 13).

When the first-formed conidium is mature, it is pushed to one side by the formation of a second at the same point, and so on until five or more are produced (Fig. 167, 8). These remain adhering by surface attraction, unless roughly handled or immersed in water, when they become free. They can germinate within 24 hours, giving usually a single terminal germ-tube, though the septate spores may have one from each end. The germ-tubes of adjacent spores often unite (Fig. 167, 12).

Infection of cane by this fungus takes place very much as in *Colletotrichum falcatum*. Inoculation of healthy stems can be done through wounds which expose the pith, through leaf scars, and through the adventitious roots and root "eyes" at the nodes. The progress of the infection within the stem is slow, only 4 to 6 internodes having been reached after three months in some experiments where the fungus was introduced through a hole into the pith. The uninjured rind cannot be penetrated. Cut setts have been infected and the resulting shoots killed by the disease. It is probable that wound infection is far more common than in red rot. No case of leaf attack has been observed in the field, but laboratory experiments prove that the leaf can be attacked (Fig. 167, 1), the infection resulting in a leaf spot resembling that caused by *Helminthosporium Sacchari* (p. 406).

This fungus frequently occurs with *Colletotrichum*, causing a mixed infection, but the reason for their co-existence in so many cases is not known. It is probably present in the soil of cane fields, as the genus to which it belongs contains many soil dwellers. Infection of the planted setts in artificial inoculations causes many deaths within the first three months of growth, but as this is not commonly noticed in natural attacks, the conclusion is that soil infection does not often occur, wound infections above ground being probably the usual mode of attack.

The control of the disease should be on the same lines as in red rot (see p. 399). As, however, wound infection is far more common, the importance of removing diseased clumps before they have time to rot and set free spores is much greater. As a rule, the disease is not a severe one, spread within the cane is gradual, and communication from one plant to another slow; and though our experience with it is limited as yet, it is probable that it is incapable of doing permanent damage, so long as the measures advocated against red rot are carried out.

**Brown leaf spot** (*Cercospora longipes* Butl.).—This is by far the commonest leaf disease of cane in India, though it does not appear to be

known in other countries. It is practically confined to the thin canes, in contradistinction to the ring-spot disease, which is usually found only on thick kinds. It is interesting to recall that certain Java authorities have suggested that the thin and thick canes of India may have been derived from different species of wild *Saccharums*, so widely are they separated from one another.

Over most of northern India, it is rare to find a field of the ordinarily cultivated varieties free from the brown leaf spot, and it is often so intense as to impart a distinctly reddish tinge to the foliage. Like other leaf diseases, it does not kill the cane, but the injury to the leaf surface must considerably reduce the yield of sugar where it is severe.

The spots are confined to the leaf blade and do not occur on the leaf sheath. They appear when the leaves are not yet fully grown, and continue until they fade. At first they are narrow, oval, about one-eighth of an inch in length, reddish in colour, and visible on both surfaces of the leaf. They increase in size, and a brown centre early becomes evident. At the same time, the tissue around the spot becomes discoloured and a well-marked yellow areola may be formed. At this stage, the spot is tricolour, brown, red, and yellow zones being formed concentrically. As it widens, however, the brown portion increases at the expense of the others, and the centre dries up and loses its colour. The spot now consists of a broad, oval, deep-brown ring, with a straw-coloured centre where the leaf has withered. These rings may reach a size of half an inch in length by an eight or more in breadth. They may run together to form larger groups. The brown colour persists even after the leaf has fallen.

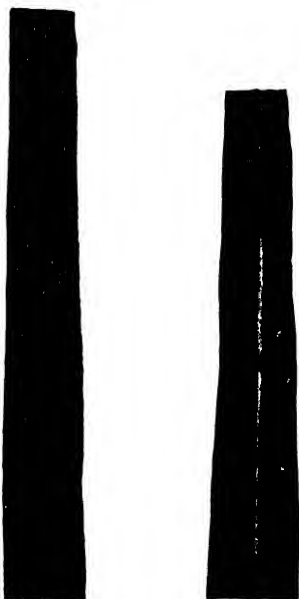


FIG. 168. Brown leaf spot of sugarcane (*Cercospora longipes*): early stage on left, later stage on right,  $\times \frac{1}{2}$ .

At the centre of the spot, the thickness of the leaf is diminished. Here, fungal hyphae are found ramifying on the under surface, external to the epidermis. They also occur, but less often, on the upper surface. The cells of the epidermis are corroded and those of the leaf mesophyll, between the bundles, much discoloured. This browning of the cells is produced although the hyphae do not penetrate within them, and is perhaps due to the secretion of some protoplasmic poison capable of traversing the cell walls. The cell contents are early killed and collected into a brown mass at the centre. Cells without living contents, as the vessels and fibres, and also the thick-walled sclerenchyma of the leaf, are not affected. At a later stage, rather large, brown hyphae force their way between the disorganised cells to the interior of the leaf, and narrow cracks containing hyphae are sometimes visible as a result of this.

Spores are formed soon after the brown centre makes its appearance in the spots (Fig. 11, 4). They are borne chiefly on the under surface, rarely above. Numerous clusters of long, brown, unbranched, septate, abruptly bent or "kneed" conidiophores arise at right angles to the surface from the centre of the spot. They form an elongated velvety cushion visible with a lens. On them are borne elongated conidia in the manner already described as general in the genus *Cercospora*. The conidiophores are flexible and measure 100 to 200 by  $4\mu$ , while the spores are hyaline, broader below, attenuated above, straight or curved, with 4 to 6 septa, and measure 40 to 80 by  $5\mu$  in diameter.

At a later stage, a second type of fructification is found, as little sharp, black dots, arranged in rows, after the centre of the spot has dried up. They are spherical, composed of polygonal cells, furnished on the surface with blunt, septate processes, and are probably spore cases, though they remain sterile for months and have never been found to contain spores. They may be a resting form of fructification like the perithecia of some of the mildews (c.f. *Erysiphe graminis*), but have not been proved to belong to the conidial fungus, though their constant succession on the same spots and apparently from the same mycelium, makes this probable.

Direct treatment of this disease, as by spraying, is scarcely to be thought of. There seems as little likelihood of treating it effectively as in the cereal rusts. Possibly resistant varieties may be found or produced by breeding, or further study may show that cultural methods can reduce the susceptibility of the cane, though at Pusa it appears every year irrespective of the treatment of the cane.

**Helminthosporiose** (*Helminthosporium Sacchari* Butl.).—The leaves of sugarcane are sometimes attacked at Pusa by a species of this common genus of leaf parasites. It has not been observed in other parts of India, but has probably been mistaken for some of the other leaf spots and so escaped notice. A closely related fungus is known in Java and Hawaii, under the name of *Cercospora Sacchari* (which appears to be more properly a *Helminthosporium*), causing a disease known as "eye spot."

The infected leaves first show small red spots, which enlarge rapidly, chiefly in a longitudinal direction and, especially towards the tip of the leaf, may run together to form long streaks. The centre of the spot soon changes to a dirty straw colour, around which the margin remains red for a time and then becomes dark brown (Fig. 162). The spots occur equally on the thin part of the leaf and on the midrib, where they may be taken

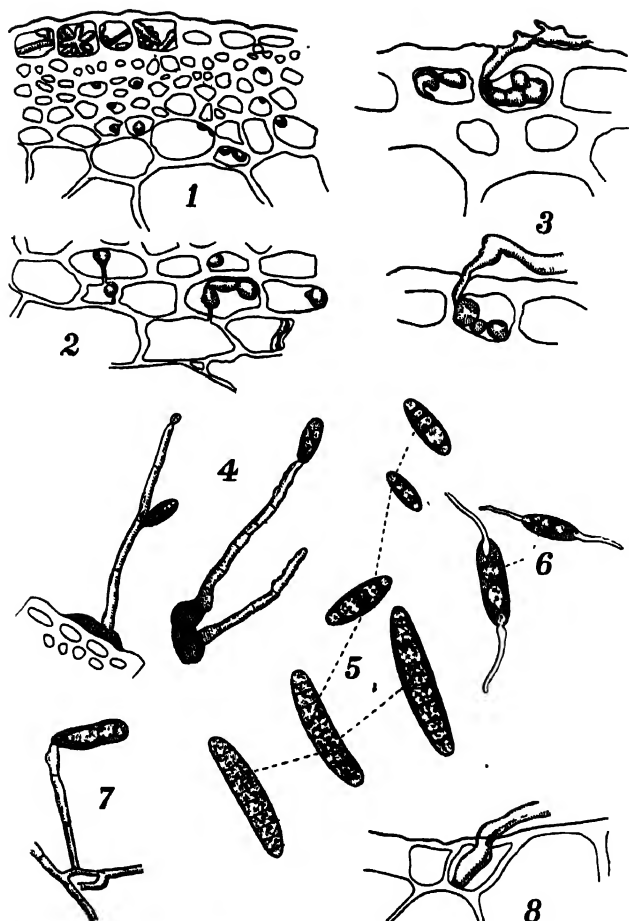


FIG. 169. *Helminthosporium Sacchari*. 1, hyphae in outer cells of leaf,  $\times 260$ ; 2, hyphae penetrating cell walls,  $\times 375$ ; 3, entry of hyphae into epidermal cells,  $\times 700$ ; 4, conidiophores with young conidia,  $\times 260$ ; 5, conidia, those above immature,  $\times 375$ ; 6, germination,  $\times 375$ ; 7, small conidiophore and conidium from culture,  $\times 260$ ; 8, base of conidiophore arising within the epidermis,  $\times 375$ .

for the midrib form of *Colletotrichum falcatum*. When numerous, they cause death of the leaf tissues beyond the limits of the spots; the tip of the leaf often withers completely, and there may be long, withered strips down the margins.

The mycelium of the parasite is found in the leaf cells of the spotted portion, and also collects in small stromatic masses on the surface of the spot. The hyphæ are brown near the surface, but hyaline deeper in. They pass from cell to cell through narrow cracks in the walls, which are especially noticeable in the thick-walled sclerenchyma (Fig. 169, 2); within the cells they are swollen so as almost to fill the cavity of the smaller cells. In the epidermis, they frequently form small stromatic masses (Fig. 169, 1). The cells appear to be killed in advance of the growth of the fungus, as although the hyphæ are numerous in the dead cells, it is rare to find penetration of a still living cell.

As soon as the centre of the spot begins to turn straw-coloured, fructification occurs by the growth of sporophores from the stromata, both those within the epidermal cells and those on the surface of the leaf. The sporophores are stout, erect, rather rigid hyphæ, arising from the outer cells of the stroma (Fig. 169, 4). They are usually unbranched, 3 to 10-septate, dark greenish-brown below, paler above, and with several angular inequalities, each marking the point of insertion of an originally apical spore, beyond which the sporophore has continued its growth. They are 100 to 190 by 5.5 to 7.5  $\mu$  in diameter.

The spores are borne singly and readily fall off. They are cylindrical or long elliptical in shape, with very thick walls, olive-green to brown in colour, 3 to 10-septate, and 35 to 60 by 8.5 to 12  $\mu$  in diameter (Fig. 169, 5). They germinate very quickly by a germ-tube from each end (Fig. 169, 6), giving a much branched, closely septate mycelium (Fig. 2).

Healthy, unwounded leaves can be readily infected by sowing the spores on them in a moist atmosphere. Penetration occurs directly into the epidermal cells (Fig. 169, 3) from which some hyphæ pass to the deeper tissues while others collect into small stromata. The pressure from these is sometimes enough to rupture the outer walls even before fructification begins. The latter is usually confined to the quite dead central part of the spot.

Little requires to be said regarding the treatment as, so far, this disease has not been found to damage the crop materially. If it became severe, it might be advisable to strip off and destroy the affected leaves in the early stages of the attack (it has been found as early as June, in Pusa). But it seems unlikely that sufficient damage can be caused by it to make any direct treatment profitable.

**Banded sclerotial disease (sterile mycelium).**—No other leaf disease of sugarcane is likely to be confused with this, as it causes a very characteristic discoloration of the affected parts. It is not common in India, but has been seen at Pusa, Jorhat, and in parts of South India. Like most of our cane diseases, it was first described in Java, where it is widely distributed but seldom of great intensity. It does not seem to be known elsewhere.

The attack is usually confined to the lower part of the leaf, but at times involves also the leaf sheath. It begins as a discoloured patch, at first dirty green, then liver-coloured, and finally yellowish to white,

visible on both surfaces of the leaf. The patch is of very irregular shape but often tends to be broader than long, and is always sharply delimited by a narrow, purple or red-brown zone. A number of such patches usually succeed each other along the leaf, separated by narrow, green or brown gaps, the result being a variegated, banded appearance which is very striking. At first the midrib is not attacked, but at a later stage it is often invaded by the growing patches. The limitation to half the leaf breadth is, however, on the whole, fairly constant. The banded succession of spots is perhaps due to the parasite ceasing to extend along the leaf beyond each newly invaded area, until after it has entered the tissues and exhausted the food obtainable therein. The fungus seems in this way to have alternate periods of extension and nutrition.

Usually several successive leaves are attacked, the disease occasionally reaching even up to the growing shoot. On the sheaths, the growth is slower and the colour changes less marked than on the leaf blade. The stem seems never to be attacked.

On close examination, especially in damp weather, a fine, white, cobweb-like fungus growth can be seen on both surfaces of the leaf, most easily on the still green part beyond the leaf spot, and on the under side. If a bit of leaf thus affected be placed on a sound sugarcane leaf in a moist atmosphere, the white growth rapidly extends to the new leaf and soon begins to form characteristic spots. Later, little condensations of fungus tissue can be noticed commencing to form on the under surface, and these develop into small, hard, sterile masses, the sclerotia, which are from 1 to 3 mm. in diameter, at first white, then brown, and rather irregular in shape (Fig. 170, 2).

The superficial mycelium is white and silky. It is composed of a layer of thick hyphae, not much septate or branched at first, then branching and anastomosing freely, about  $8\mu$  in maximum diameter, and closely applied to the leaf surface. Side branches enter the stomata (Fig. 170, 4), and reach the leaf parenchyma, where they grow first



FIG. 170. Banded sclerotial disease of sugarcane. 1, affected leaf bearing sclerotia,  $\times 4$ ; 2, sclerotia; 3, section of part of sclerotium,  $\times 120$ ; 4, infection through stoma of leaf,  $\times 275$ . (After Lucassen and Wakker.)

in the intercellular spaces, but later enter the cells. Occasionally, at a late stage, the vessels are penetrated; this seems to be more frequent in the sheath than in the leaf blade. The cell walls are turned red at an early stage; the colour then gradually fades, and at the same time the chlorophyll loses its green tint and the whole tissue becomes brown. The brightly coloured marginal band round the spot thus represents the area into which the hyphae have recently extended.

The sclerotia are found on the under surface of the dead spots, attached to the mycelial layer at first, but afterwards free as the latter disappears. They are composed of brown-walled pseudoparenchyma of a uniform nature, with no differentiation into cortex and medulla such as is found in many bodies of this nature, except for a slightly darker colour of the walls near the surface (Fig. 170, 3). The latter is dull and with a few loose hyphae. In culture, sclerotia may form in six days. No other stage is known, and spore formation, if it occurs, cannot be common. At the same time the characters of the mycelium and sclerotia permit the guess that the fungus will be found to belong to the parasitic group of *Hypochnus* or *Corticium*, represented in India by such forms as *Hypochnus Solani* and *Corticium Koleroga*; and this idea has already been suggested in Japan, where it is thought that the present fungus may be the same as *Hypochnus* (*Corticium*) *Sasakii* Shirai a Japanese species found on camphor. *Sclerotium Oryzae* Catt., which has also been included in the same species, is certainly different from the sugarcane parasite, though both can attack rice.

Infection is brought about largely by means of the sclerotia, which can preserve their vitality for a long time if kept dry, and renew growth when again moistened. This, no doubt, usually happens from the soil into which sclerotia from a previous crop have been shed. The sugarcane leaves are then infected by direct contact with the soil, or by growth of the mycelium up the lowermost leaf sheath, or, finally, by growth first up some of the wild grasses or weeds under the cane until a cane leaf is encountered. The latter is said to happen frequently in Java, where also stools with shoots near the ground, and leaves liable to touch the soil, are often affected. Growth is most vigorous in moist weather, the disease being one closely dependent on humidity.

Though the damage caused by this fungus to an individual leaf is greater than in, perhaps, any other leaf disease of the crop, yet, owing to its imperfect means of spread, the actual loss caused by it is usually small. The most serious form is when the young shoots are reached. On the lower leaves, the injury affects the cane but little.

The only treatment is to remove and burn early all infected leaves before the sclerotia are shed. It is necessary to remove all withered leaf parts that may have been reached by the fungus, as the sclerotia form most readily on dead parts. If the soil is likely to have many sclerotia from a previously diseased crop, clean weeding and early stripping of the lower leaves would probably diminish the risk of infection. In the ordinary sporadic cases, no treatment is required.



The fungus is known on rice, three wild grasses (*Saccharum spontaneum*, *S. arundinaceum*, and *Rottboellia exaltata*), and arrowroot (*Maranta*), and can infect sugarcane from these plants. There are probably other hosts.

**Sooty mould** (*Capnodium* sp.).—It is not uncommon to find leaves, or even whole plants, of sugarcane turned black by the deposit of a superficial black mould. Such plants are always found to be infested with insects (such as *Aleurodes barodensis* Mask.) which are capable of producing the secretion known as "honey dew." In some cases, this substance is formed in such quantity as to drip from leaf to leaf, and even on to grass or weeds under the cane. Wherever it is found, the fungus accompanies it, but as soon as its production ceases, or the insects leave the plant, the mould gradually flakes off and disappears.

The black covering is purely superficial, and is not in any sense parasitic; its presence is nevertheless harmful, in that it bleaches the leaf under it by cutting off a certain amount of light, without which the green chlorophyll of the leaf is unable to develop or to elaborate food. The insects of whose presence it is a sign are mostly very harmful, so that it is not to be wondered at that the cultivators regard the black fungus (whose connection with insects is probably not suspected) as one of the worst diseases of the crop.

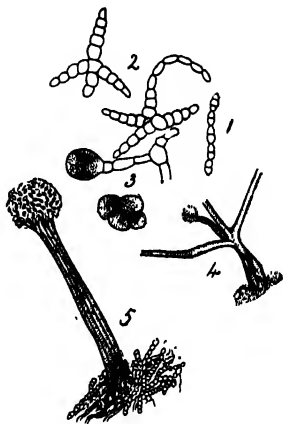


FIG. 171. Sooty mould of sugarcane: 1, segmented hyphae; 2, *Triposporium* form,  $\times 375$ ; 3, *Coniothecium* form,  $\times 375$ ; 4, branched pycnidium,  $\times 55$ ; 5, a pycnidium with spores,  $\times 175$ .

The black crust is composed of branching, septate, brown hyphae, which tend to break up into mostly 2-celled, oval segments, 12 by  $9\mu$  in diameter (Fig. 171, 1). Attached by short stalks to some of the hyphae are blackish masses of cells, composed of 2 or more cells, closely united into a more or less cubical mass. This is a structure characteristic of the form-genus *Coniothecium* (Fig. 171, 3). Another conidial form occurs sometimes in great quantity, the spores being composed of several cells arranged along 4 (occasionally 3 or 5) radii and tapering towards their free ends. The radii are mostly 4-septate and measure 20 to 33 by  $8\mu$ . This type is that of the genus *Triposporium* (Fig. 171, 2). Finally, in many cases, numerous pycnidia are produced. These are very variable in size

and shape, but agree in being slender, elongated, composed of deeply coloured, septate hyphæ, and opening at the top by a fringed mouth. They are often branched, and are cylindrical but often with irregular enlargements (Fig. 171, 4). Within them are quantities of small, 1-celled, hyaline spores,  $4\frac{1}{2}$  to 5 by 2 to  $3\mu$  in diameter (Fig. 171, 5). Perithecia have not been found, so that it is not definitely known to which of the several genera of the sooty moulds the fungus belongs, and it is only provisionally referred to *Capnodium*.

Treatment should be confined to getting rid of the insects, which are the direct cause of the trouble.

## CHAPTER XII.

### TEA.

(*Camellia Thea* Link.)

THE diseases of tea are exceedingly numerous, all parts of the plant, leaves, stem, and roots, being subject to the attacks of various parasites, especially in the older gardens. They are by no means fully investigated, and in many cases our information is scanty and of doubtful reliability. As an instance of the difficulties met with, it is sufficient to mention that the fungus called *Pestalozzia palmarum* (or *Guepinii*), which has for many years been considered the cause of a serious disease known as "grey blight" in India, is stated in Ceylon to occur everywhere in tea and to do so little damage that no steps are taken to combat it: and both the above names for the species are incorrect. Further, though a large number of root diseases of different kinds is known, the cause has not yet been definitely established in a single case. There is naturally a strong presumption that the fungus found to be invariably present in each case is the cause of the disease, but the proof, which requires that the disease can be artificially produced, has not yet been furnished. Indeed, scarcely any of the diseases of tea, with the exception of red rust and blister blight, have been investigated from this point of view, and we know little of the life-history of the organisms associated with them and presumed to be their cause. Hence, though there is strong reason to believe that such measures to check the ravages of fungal pests as spraying will ultimately be forced into use by the pressure of competition and the increasing losses from this cause, it is exceedingly difficult to advise as to the best methods to adopt, because of the lack of detailed knowledge of the life-history of the parasites and of the economic and other considerations which have to be taken into account when spraying on a large scale is undertaken.

**Red rust** (*Cephaleuros Mycoidea* Karst.).—All the diseases of plants of cryptogamic origin, hitherto considered, have been caused by parasites of the classes of fungi and bacteria. That now to be discussed is, therefore, of particular interest, as being one of the rare cases produced by members of the class of algæ, of which our most familiar examples

are the seaweeds and alime-weeds of salt and fresh water. It is further of interest as being, in the opinion of some of our best observers, the most serious blight caused by cryptogams to which tea is subject in north-east India. It is found in quantity sufficient to cause serious damage, where the condition of the tea bushes is such as to favour its presence, throughout Assam, in Chittagong, in the Duars and Terai, and in the lower Darjeeling hill districts; that is to say in by far the larger and more important of the tea areas of India. In the higher parts of the Darjeeling tea districts, in Dehra Dun, Kumaon, and the South Indian hills, it has not hitherto been found to cause appreciable loss. Outside India, too, it appears to be of minor importance, though the alga is widely distributed throughout tropical Asia, Africa, and America.

Besides tea, it occurs on a host of other plants: in the Calcutta Botanic Gardens alone, on some twenty species belonging to the most diverse families, from ferns to forest trees and garden flowers. A complete list of its hosts in India would probably exceed a hundred, and it is somewhat remarkable that, so far, only one other plant of economic importance, the mango, has been found to suffer from its attack in this country. Liberian coffee is, however, sometimes injured by a very similar, or even, probably, identical alga in Java and elsewhere, and recently it has been found that *Cephaleuros Mycoidea* attacks cacao twigs in the Belgian Congo, producing a serious disease of the same type as that in tea.

On many of the plants on which *Cephaleuros Mycoidea* has been found, it occurs as a simple epiphyte on the leaves, growing on the surface only and not penetrating the leaf tissues; this is the usual characteristic of the members of the Chroolepidæ, to which family of algæ *Cephaleuros* belongs. Not infrequently, however, the present species penetrates the leaf cuticle and extends between the cuticle and epidermal cells; this has been observed in coffee and some other plants. Sometimes the penetration of the leaf is more complete, the alga passing down between the cells of the parenchyma and even reaching from one surface of the leaf to the other; examples are found on tea and (very commonly) on *Cinnamomum iners*. The form on coffee in Java occurs on the young berries as well as the leaves, some of the filaments penetrating the tissues of the fruit, though the majority remain superficial. A similar condition has been found on the tea fruit in India. Finally, in its most serious form, the alga attacks the young woody shoots, which become cankered and are killed. It is only in this form that it is really a destructive

parasite, tea and mango being the most completely studied cases, though the stems of other plants are not infrequently attacked.

On tea, therefore, we must distinguish between two easily recognisable forms of red rust: on the leaves, where it is sometimes epiphytic, sometimes parasitic; and on the stems, where it is ordinarily parasitic. The leaf form can be found throughout the year in an active condition. The stem form, on the other hand, easily escapes notice except at certain periods. It is usual in north-east India to prune practically all the tea between November and March, the recently formed wood of the past season being cut back to from 1 to 4 inches. The characteristic fructifying patches of red rust are never found during this period on the young wood, though an occasional patch may be seen on the older wood. About the end of March or April, however, generally after the first heavy rain of the season, the remnant of the previous year's wood not removed in pruning is often found covered with red, hairy patches (Fig. 173, 1). The new shoots arising from this wood will, at the same time, be noticed to show a lack of vigour, or even to cease growth prematurely; instead of remaining green and succulent, they rapidly become covered with bark almost up to the tip, and their leaves show, often in most striking fashion, a condition of chlorosis indicated by the green colour fading more or less completely to white. Such shoots usually die off gradually, it being comparatively rare for a shoot which bears algal patches in any quantity to recover. If they do so, the signs remain on the stem as a cankered patch, which may be up to about three-quarters of an inch in diameter.

The red, hairy patches characteristic of the fructifying condition of the alga on the wood are found in greatest quantity from April to June, but they continue to occur right on until the end of the year. Usually, as just described, they occur on the stems of the second year of growth, but similar patches can sometimes be found on older wood and even on the main stem. The older wood is, however, more resistant, and the attack does not seem to cause its death beyond the limits of the algal patch, as is sooner or later the case when it is found on second year's wood. The number of fruiting patches becomes smaller as the season progresses, from June onward, until, by November, it is usually difficult to find such a patch. This does not mean that the parasite is not there but merely that it is not actively fructifying.

The leaf form of red rust does little damage, and is of little significance, except in so far as it serves as a source of infection to the stems.

Its prevalence is not affected by the healthiness of the bush ; it can be found in almost any garden, and often on almost every bush. The alga occurs as orange-yellow, roughly circular patches on the upper surface of the leaf. The patches may be numerous or few, crowded or scattered, and may occupy any part of the leaf, even the midrib, though they are rare on the petiole. Their maximum diameter is up to about one



FIG. 172. Red rust of tea (*Cephaenurus Myrsinæ*) : leaves with red rust.

centimetre. When fruiting, they are covered with a dense mass of delicate, erect, orange-coloured filaments, the stalks of the sporangia. In the earlier stages, and often throughout the development, the patch is raised slightly above the surface of the leaf, but in older cases, when penetration of the leaf occurs, the centre may be depressed owing to the death and collapse of the leaf cells, while the margin may remain as a raised ring.

The thallus of the alga appears, under the microscope, as a disk of radially elongated cells, arising by centrifugal growth and repeated dichotomous division from the germinating spore. In vertical section, it is seen that the disk is composed of more than one layer, the greatest thickness being in the centre. It may be wholly external to the surface of the leaf or may be partly sunk in the cuticle, as the germinating spore seems to possess the power sometimes of embedding itself firmly in the cuticle, and as soon as active vegetative growth occurs, the algal cells, in many cases, are found pushing their way beneath the cuticle as well as above it. The epidermal cells and the palisade parenchyma immediately below the alga are generally discoloured and dead, the palisade cells frequently showing sub-division by transverse septa as the result of induced meristem formation. Sometimes a layer of cork is thus formed, which prevents the penetration of the alga, particularly towards the margin of the patch; and the swollen margin found often in old patches is produced in this manner. In other cases, there is little obvious alteration of the tissues under the algal growth, though the conditions leading to these different effects are not understood.

Up to about August, in normal seasons in Assam, penetration beyond the leaf cuticle does not usually occur. Later on, or, if there is excessive rain, earlier, many patches are found completely penetrating the leaf and producing a corresponding growth on the under surface (Fig. 173, 2). When this occurs, the patch on the upper surface generally exhibits an annular formation. The erect hairs are missing in the centre, where the algal cells are now mostly dead. Many of these cells are filled with air, which gives them a lustrous, white appearance, apt to be mistaken on casual examination for a lichenous growth. The penetrating filaments of the parasite may extend laterally between the epidermis and the subjacent layers for a considerable distance all round, while certain of them force their way vertically downwards between the palisade cells. The algal cells are sometimes difficult to distinguish from those of the host, though they are usually marked by the presence of the orange-yellow colouring matter "hæmatochrom," characteristic of *Cephaleuros* and its allies. The filaments never penetrate the leaf cells, but get nourishment by osmosis. The cells in contact are killed, and their contents turn brown and dry up. Ultimately, the alga itself ceases growth, its cells die, and a crater-like depression is left, surrounded by an elevated ring.

The death of the alga is sometimes hastened by an invasion of fungal hyphæ, which are found very frequently intermingled with the thallus. Some observers have considered that lichen-formation is brought about by this invasion, the two organisms living in association, but it seems more probable that the fungus is wholly injurious and, from the first, an enemy of the alga.

Quite different is the course of events when the stem is attacked. In tea, the first-formed shoots are smooth and green, but during their subsequent growth they become covered with an irregular bark, which, in the second year, becomes smooth and remains so. It is, in the great majority of cases, only during the period of irregular bark that infection with red rust occurs. The young bark of tea is not formed in a uniform layer. Læunæ exist between patches of cork-forming cells, and the surface layers are exfoliated in an irregular manner, leaving gaps unprotected by cork for a time. In a healthy shoot, such gaps are rapidly filled up, and any spores which may have lodged in them are thrown off with the outer bark layers. Any conditions which weaken the growth of the bush delay this process and increase the danger of the alga getting a successful footing and reaching the inner layers. While the shoot is green, infection seems to be rare, and again when the bark formation has become uniform, and the definite smooth bark of the tea stem has made its appearance, few channels are offered for attack. That such channels do sometimes exist is evidenced by the occasional occurrence of the alga on the old wood, as already mentioned.

The examination of the young shoots shows that small algal patches occur frequently in the crevices of the bark. Here, if conditions are favourable, the algal cells are found

pushing their way among the cells below the bark and producing, by their growth, profound disruptive effects on the tissues of the host (Fig. 173, 3). Unlike what is found in leaf attacks, the internal cells in the stem are usually green and without the orange-yellow haematochrom already described as characteristic of the alga. The cells of the cortex in the neighbourhood of the ingrowth are killed. Sometimes this serves to cut off supplies of moisture and nourishment from the alga and leads to cessation of its growth; sometimes the algal cells develop too fast to be thus checked, and spread past the resistant layer. The result in either case is the removal of successive layers of bark, and, where this fails in throwing off the parasite, the latter penetrates ever deeper into the cortex and

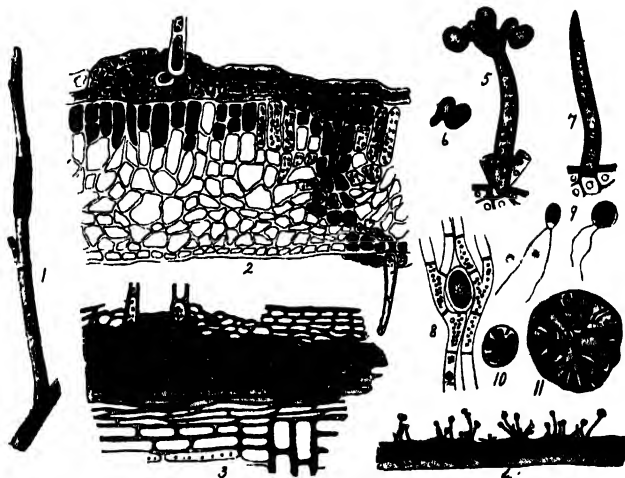


FIG. 173. Red rust of tea (*Oophialeurois Mycolidea*): 1, shoot with rusted patch near middle and healed scar below; 2, section showing penetration of alga through leaf; 3, section showing alga penetrating bark of stem; 4, fruiting stage on stem, enlarged and diagrammatic; 5, fertile hair bearing sporangium; 6, a sporangium with its pedicel; 7, sterile hair; 8, sessile sporangium in the thallus,  $\times 180$ ; 9, saccus, ovoid,  $\times 420$ , and round,  $\times 490$ ; 10, germination of the saccus,  $\times 960$ ; 11, young disk from a saccus,  $\times 350$ . (1-4 after Mann and Hutchinson, 5-7 after Delacroix, 8-11 after Cunningham.)

may ultimately cause the death of the shoot. In the early stages, the algal growth is small, but later it may spread up, down, and round the affected shoot, so that, in aggravated cases, the latter may be completely invested for several inches of its length. When completely surrounded, the shoot is found either moribund or, frequently, quite dead for some distance behind the seat of infection.

In an unhealthy or non-vigorous bush, the thin, straggling shoots, which are more common near the periphery, are the worst nourished, and, as might be expected, are the first to be attacked. Similarly in unpruned bushes, a large number of the young stems are liable to be thin



and ill-developed, owing to their great distance from the source of food supply. Experiments show that such bushes are much more liable to attack than pruned tea. It is probable that infection usually takes place in the rainy season (July to October or November), but unless examined microscopically, the attack is not observed until the following March to May, when the fructifications first appear. By the end of the year, in bad attacks, the bushes will often be found to be surrounded by a ring of dead twigs, and to have a certain number of dead twigs through-

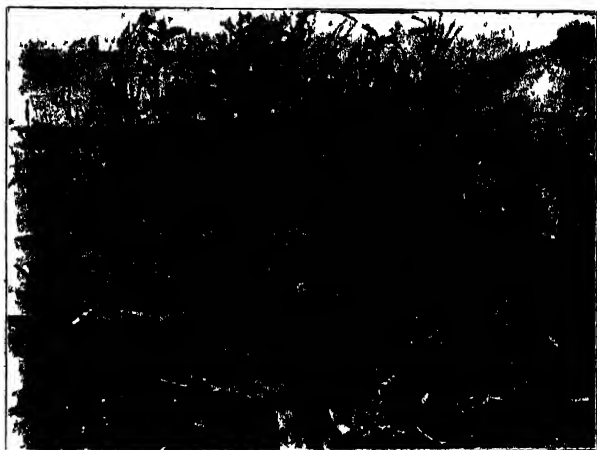


FIG. 174. Red rust of tea (*Cephaleuros Mycoides*) : seriously damaged bush.

out, while a few of the stronger shoots in the centre of the bush have alone been able to throw off the blight, and are growing vigorously. This appearance, coupled with a general aspect of hollowness, due to death of so many twigs, and the yellow, unhealthy colour of most of the leaves, renders the recognition of a severe attack quite an easy matter after a little experience, even in the absence of any red patches on the stem. The striking chlorosis of the leaves of infected shoots is most marked early in the season, but may be seen at any period of the year.

A bush thus attacked is often, if not usually, worse the following season, and the ultimate result, if the disease be not checked, is a continual reduction in size at each pruning, and ultimately death. The disease

is thus a chronic or cumulative one, and the bushes are not usually killed until after quite a number of years.

Whether on the leaf or stem, the alga, after a variable period of vegetative growth, develops its reproductive mechanism. Certain cells, either forming the termination of a radial series of vegetative cells within the thallus, or raised on vertical stalks at right angles to the surface, become sporangia. Those formed in the thallus are oval or spherical, thick-walled, about 40 to 50 $\mu$  in diameter, and with dark orange-coloured contents due to the presence of hæmatochrom, masking the green chlorophyll of the alga (Fig. 173, 8). They are formed singly, in direct continuation with a vegetative filament, and are said not to occur in the parasitic thalli on the stem. Those raised above the surface are formed on special sporangiophores, consisting of thick, rigid, septate hairs, about 1 mm. in length by 50 $\mu$  broad, swollen into a vesicle at the tip, and each carrying from 3 to 6 sporangia, which arise from the vesicle on curiously curved pedicels (Fig. 178, 5). These sporangia are oval and rather smaller than the other kind, averaging about 35 $\mu$  in length, but are otherwise similar (Fig. 173, 6). In both cases, when ripe, the contents of the sporangium segment into a number of little spherical masses, which are liberated as zoospores through an opening in the wall. The zoospores are orange-coloured, round or ovoid, and swim actively in water by means of two cilia arising near together (Fig. 173, 9). After a time they come to rest, and germinate by radial growth, accompanied by the formation of converging walls which extend from the margin in towards the centre of the spore, and divide the latter into a number of segments (Fig. 173, 10). Each segment continues to expand radially with dichotomous branching, and a new disk results (Fig. 173, 11). Some observers believe that the zoospores from the larger sessile sporangia unite in pairs before germination, and are thus sexual gametes. Besides the fertile hairs which carry sporangia, other sterile ones arise from the thallus in the same manner, recalling the paraphyses found in so many fungi (Fig. 173, 7). When the thallus is formed under the cuticle or epidermis, the hairs pierce the covering to reach the outside.

The mature aerial sporangia break off from their stalks readily. Germination only occurs in water and only during a limited period, a few days. Hence the rainy season is that best suited for propagation. On the other hand, excess of moisture checks the reproductive activity of the alga, while favouring the vegetative. The mature sporangia are also injured by excessive moisture, many bursting prematurely, without forming zoospores. Even normally-formed zoospores frequently disintegrate without germinating. Hence there are many checks to the reproduction of the parasite. On the other hand, excess of water is reduced by the hard coriaceous surface of the tea leaf, which does not retain rain drops readily, and also by the fact that the ripe alga is not easily wetted.

Dissemination is both by the wind and by water. The light sporangia are easily blown about, and they have been observed to germinate 48 hours after detachment from the stalk. Experiments have also proved that the disease may be carried from leaf to leaf and from leaf to stem with drops of water, and on certain other hosts, where the arrangement and shape of the leaves are such that the rain water follows a definite course, the distribution of the algal patches along this course is sometimes very noticeable.

The extent of the damage caused by this disease is difficult to estimate, as it is closely bound up with other conditions which reduce the vigour of the bushes. Its disastrous effects depend on whether it can penetrate the tissues of the young wood more rapidly than these are

removed by exfoliation. In other words, if the shoot is growing faster than the alga, the latter is removed and no permanent infection takes place ; if, on the other hand, the shoot is, through want of vitality, growing slowly, the alga is able to penetrate and destroy it. In some gardens in Nowgong and Cachar, it has been estimated to reduce the crop by 20 per cent., and in many places the production from old tea is less by 10 to 15 per cent. than it would be if red rust were absent. In the sandy, grey soil found in parts of the Duars, the rapid deterioration of the tea which sets in after 15 or 20 years is usually accompanied by a severe attack of this disease. Very severe attacks, leading in some cases to death of the bushes, have also been observed in the deteriorated " bheel " gardens of Sylhet.

In the control of the disease there are two considerations to be borne in mind. The parasite may be directly attacked, with a view to reducing its prevalence and consequently the chances of infection of the new wood ; and the vigour of the tea bushes may be increased, since it is established that there is a direct connection between want of vitality in the bush and the virulence of the blight.

To remove the parasite by pruning off and destroying the affected shoots, as was at one time advocated, appears to be of little use. Apart from the fact that it is very hard to remove all the growth, the new shoots again become infected, probably through the air. The tea leaves and the neighbouring jungle provide an inexhaustible reservoir of infective material. Collar pruning (*i.e.*, cutting down to the ground), and even firing badly attacked plots, are equally quite futile. It is impracticable to remove and destroy infected leaves, owing to their immense numbers.

Almost as futile have been the attempts to spray the bushes with fungicides at the time the blight is visible, that is to say, in the fruiting stage. Owing to the difficulty of wetting the patches, spraying does not sufficiently reduce the sources of infection to make it worth doing. Better results have been obtained from spraying during the winter months (the pruning season), when the parasite is usually not visible, though present in the vegetative condition. The disks are more easily wetted at this time than when they are fruiting. Bordeaux mixture of the 6-6-50 formula, applied at the rate of about 150 gallons per acre immediately after pruning has, in some cases, been markedly successful in reducing the amount of disease.

The presence of the alga on many jungle plants and shade trees near the tea, as well as on the tea itself, coupled with the probability that the

sporangia may be carried by the wind to considerable distances, combine to render largely nugatory any direct attempt to prevent the parasite reaching the new growth of the bushes each year ; it may almost be said that, provided the conditions of health of the tea are such as to favour the establishment of the blight on the wood, every bush, in many districts, will become infected.

Hence all efforts must be concentrated on increasing the vigour of growth of the bushes, so as to strengthen their capacity to resist the disease. In other words, it is necessary to find the causes of lack of luxuriance in each particular area, and to remove these. A consideration in detail of the causes which lead to defective growth of the tea bush is out of the question, as it would mean a treatise on the cultivation of tea. There is little need to do more than enumerate some of the commoner, the remedy for which is usually sufficiently obvious. The most important is probably the presence of defective soil and subsoil conditions, due to lack of drainage, to the formation of a hard pan in the soil, even merely to poverty of the soil, or to lack of adequate surface and deep cultivation. In Nowgong, for instance, it has been found that where red rust is severe, the drainage is usually defective. The system of pruning adopted also largely influences the luxuriance of the bushes, it being, of course, much safer from this point of view to obtain a smaller number of very strong, well-grown shoots, rather than a large number of more feeble ones. Hence clean pruning, that is to say, the complete removal of the small, twiggy shoots throughout the bushes, is advisable, and also the cutting of each shoot left as short as possible, consistent with leaving at least one bud in the cut stem. The effect of denuding the bush of leaves, by too close plucking in the earlier part of the season, is another extremely frequent cause of lack of luxuriance.

Finally, some types of plant are more susceptible than others, though no variety is as yet known to be altogether immune. The variety known as " Assam indigenous " is as a whole less resistant than the so-called " Manipuri " or " China " types of plant, or their hybrids with the " Assam indigenous." In existing gardens, this knowledge cannot, as a rule, be taken advantage of, as replanting is out of the question for many reasons ; but in planting out new tea, the less susceptible types should certainly be employed where the disease is prevalent and there are no strong reasons against their growth.

**Blister blight** (*Exobasidium vexans* Mass.).—The sudden and unexpected outbreak of an epidemic of this disease in the Darjeeling hill

district, where some of the best Indian tea is produced, attracted widespread attention a few years ago. The disease itself had been long known, an account of its effects on tea in Assam having been published as far back as 1868. Hitherto, however, it had been confined to a comparatively restricted area in the north-east of Assam, where in ordinary years it had not done much material damage, except in individual gardens; there had been fairly extensive outbreaks in 1895 and 1906, and a less severe one in 1900, the weather conditions in these years happening to favour the blight; but these epidemics were of short duration and the disease was not regarded as a serious or permanent menace to the tea industry, even in the affected tracts.

With the appearance of blister blight in the neighbourhood of Darjeeling, this view has had to be modified. Fears were early expressed that it would find a more congenial environment in the humid atmosphere of these hills than in its original home in Assam, and the experience of the past few years seems to have justified these fears. Since 1908, when it was first reported near Darjeeling, it has continued to spread and to take its toll of the bushes in spite of efforts to check it. It has approached the important districts at the foot of the hills and it is impossible, at the moment, to foresee the limits of its extension. The mass of the tea districts in the middle and lower parts of the Brahmaputra Valley, in Cachar, Sylhet, the Duars and Terai, are still free, and, as will be seen, there is ground for hope that climatic conditions will prevent it ever getting a permanent hold in much of this area. Meanwhile it is an actual cause of considerable loss in the Darjeeling hills, and a source of potential danger to certain other important districts. It is not known in any other tea-growing country, if we except a recent report from Formosa.

Blister blight attacks tea chiefly during the rainy season, from June onwards, in Darjeeling. In Assam, it appears at the end of April or in May. Early attacks (as early as January in Assam and in March in Darjeeling), have been reported in some years, and occasional new blisters, bearing living spores, can be found practically throughout the cold weather, but after November are rare.

The first symptom is the appearance of small, pale or pinkish spots on the leaves. These spots are round from the first, and rapidly enlarge to a diameter of from  $\frac{1}{4}$  to  $\frac{3}{4}$  of an inch, or, rarely, to almost an inch. The colour sometimes becomes deep red on both sides of the leaf. More usually, the upper surface is pale green, yellowish, or pinkish, and shiny

as if varnished, while the under surface is dull and becomes first grey and powdery and then pure white and downy. On the upper side of the leaf, the spot becomes depressed into a shallow cavity, the under side bulging out into a corresponding blister-like swelling. Sometimes this shape is reversed, the blister appearing on the upper surface and the hollow below. Both conditions may be found on the same leaf, but the white surface, which is due to the outgrowth of the fungus in its fructifying stage, is always most evident below. In old stages, the blister turns dark brown and shrinks to a flattened patch. The buds are attacked as well as the young leaves, but leaves over 4 weeks old are immune. The number of blisters on a leaf may be up to about twenty. On the mid-rib they lose their circular shape, extending more along the rib than across it; in this position, and also when the blisters are near the apex or margin or when several coalesce, much distortion and curling of the leaf may be caused.



FIG. 175. Blister blight of tea (*Exobasidium vexans*): under side of blistered leaf.

After the first appearance on the leaves, the disease spreads to the petioles and the young, succulent, green stems, where, though not so conspicuous, it causes more serious damage. No blister is formed on the stem, and the spot early becomes elongated along the stem, afterwards gradually extending round it. A red colour is not developed, and the fructifying stage is often grey instead of white. The affected tissues are swollen somewhat. Gradually the parasite eats into the stem, so that the leaves and buds above wither and blacken and the stalk bends over at the affected point. Since the attack develops usually at the time of the first flush, the young shoots are killed when they have given two or three young leaves. This means that the new growth is largely ruined, and the bushes have to make a fresh start right from the level of pruning. While there is no evidence that heavily pruned tea is attacked more virulently than lightly pruned or unpruned bushes, the damage done is much greater in the former, as the new growth in heavily cut-back tea is relied on to form a vigorous framework of good bearing wood for productiveness in subsequent seasons. The destruction of leaves and

shoots in the new growth of such bushes seriously injures the bush for several years. In other cases, the loss is chiefly to the yield in the current year, both by actual loss of leaf, and (when the attack is early) by reduction of the vitality of the bush by interference with the functions of the leaves in elaborating food, and consequent retardation of the normal healthy flushing throughout the season. In very severe attacks, it is probable that the bush is permanently injured, owing to lack of nourishment of the shoots leading to the formation of weak, twiggy wood.

The course of an attack in a garden is often very erratic. Individual bushes may be heavily attacked, others lightly, others again remain free from infection. It does not seem to begin in one place (though in Assam unpruned bushes seem to serve as foci of infection) but to develop erratically all over the block at the same time, especially when there is a spell of wet weather succeeding a few days' sunshine. It may cease almost as suddenly, and in Assam usually does so as soon as a period of hot, fine, sunny weather sets in. The rate of dissemination is rapid, and the influence of prevailing winds in causing spread in definite directions sometimes quite noticeable. It is said that a block affected one year may be almost free the next, even when the disease is prevalent in the neighbourhood. It is, therefore, not a cumulative disease like the red rust, the difference being sufficiently explained by differences in the life-history of the two parasites.

The mycelium of the parasite in the leaves is confined to the blistered areas, where the hyphae are found ramifying between the leaf cells. The green colour of the cells is reduced, the starch disappears, and the cell sap becomes pinkish, giving the reddish colour to the spot already mentioned. The cells of the spongy parenchyma usually increase in number more rapidly than do those of the palisade layers, and this uneven development causes the bulging growth on the under side of the spot. This is increased by an accumulation of hyphae on the inside of the lower epidermis, which push apart the outer layers of the spongy parenchyma (Fig. 176, 6-7).

The hyphae are very fine,  $1.5$  to  $2.3\mu$  in diameter, septate, and with a tendency to collect in bundles. After a time, little groups of them emerge through the stomata and bear spores. The hyphal masses below the epidermis increase at the same moment and soon rupture the epidermis by their pressure, so that an almost continuous layer of vertical hyphae appears over the surface of the spot, causing the grey or white coating already mentioned (Fig. 176, 1). When spore-production is vigorous, the surface of this layer becomes powdery.

The spores are of two kinds. By far the most abundant are conidia, borne singly at the end of long stalks. They are hyaline, elliptical, sometimes unseptate but usually with a median septum when mature, straight or lightly curved, and measure  $12$  to  $21\mu$  by  $4.5$  to  $6\mu$  in diameter. Conidia with two septa are sometimes found. They germinate after a few hours in water, giving a germ-tube from the end (Fig. 176, 2-3).

The second spore form is that characteristic of the Basidiomycetes, to which group the parasite of blister blight belongs. Basidia are formed in the surface layer in fairly

large numbers, but never so as to make a continuous hymenium. They are intermingled with the conidial stalks and sterile hairs. Each basidium is long, club-shaped, and with

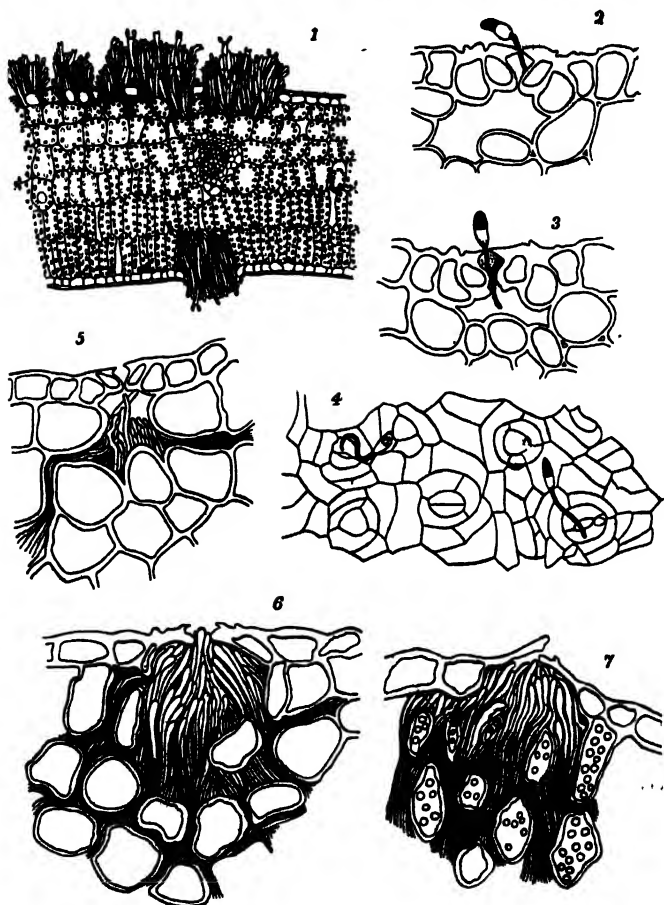


FIG. 176. *Eoobasidium vesans*: 1, section of the leaf showing fertile hyphae of young blisters,  $\times 145$ ; 2-3, germ-tubes of conidia infecting through stomata,  $\times 680$ ; 4, same, under surface of leaf,  $\times 440$ ; 5, hyphae accumulating under a stoma,  $\times 440$ ; 6, same showing bursting of stomata,  $\times 440$ ; 7, hyphae bursting through epidermal cells,  $\times 440$ .

usually two short sterigmata at the end, each bearing a basidiospore (Fig. 176, 1). The basidia are from 30 up to 90 $\mu$  in length by 4 to 6 $\mu$  broad, the sterigmata 3 to 5 $\mu$  long, and



the spores 7 to 13.5 by 2.3 to 4.5  $\mu$  in diameter. They germinate similarly to the conidia, which they resemble except in being unseptate. Occasionally, basidia with 3 or 4 sterigmata have been observed.

Besides the fertile hyphae, others which carry no spores occur in the surface layer, and give a downy appearance to the spot when numerous. When growth is vigorous within the leaf, spore-bearing tufts may also break out on the upper surface, but they are always comparatively few in number.

Infection occurs through the stomata of the under surface of the leaves (Fig. 176, 2-4), the germ-tubes of conidia or basidiospores entering and branching into a mycelium, chiefly situated in the spongy parenchyma. The spores early lose their vitality, few surviving even the journey by post from Darjeeling to Assam (perhaps 2 to 3 days from time of picking). No resting or dormant stage is known, so that if a patch of tea can be once cleared of the living blisters, it will remain free unless re-infected from outside. The new spot is clearly visible about eleven days after infection, and six to eight days later the blister is fully formed and producing spores.

The control of the disease appears to be very difficult. The measures tried have been picking off diseased material, pruning, and spraying.

Picking off and destroying diseased leaves and shoots has been energetically carried out on some gardens, with indifferent results. In one area of 1,300 acres, over 32 tons of diseased material were gathered and destroyed in 1908 and 1909, but in August of the latter year the disease spread beyond the capacity of the available labour to cope with it. Close plucking of all shoots, whether diseased or not, repeated three times at intervals of four or five days, was tried in another garden, the idea being to remove all susceptible parts of the bushes for a period long enough to ensure that no recent infections, not yet visible to the naked eye, should be left. The new growth then developed free from disease. This was repeated with some modification on a large scale, the coolies being instructed to pick the bud and two leaves as usual, but to remove the third leaf and also the fourth if there was one. Only little shoots with a bud and one leaf were left. The garden was gone over about every eight or ten days from July, and by the middle of September there was little disease left. This method, however, is only practicable late in the season after good growth has been made, as it would be dangerous to the bushes after unfavourable growth early in the season. The labour required is also often not available.

Pruning has one important bearing on the disease. It is said definitely in Assam, and it is probable in Darjeeling also, that the blight

remains through the cold weather chiefly on unpruned tea. Certain gardens have reported that the attack appears each year at the same spot, near a seed garden, an old nursery, or a few abandoned bushes, all of which would be unpruned. In any case, the more complete the pruning, the less chance of the blight remaining throughout the cold weather. At the period of pruning, all old leaves with traces of blisters should be removed. As heavy pruning may lead to the bushes being permanently injured in a subsequent attack, severe cutting back requires to be done with much caution and avoided where an outbreak is feared.

Spraying is attended with much difficulty. It has been established that Bordeaux mixture kills the spores and superficial filaments of the parasite, and checks the spread on the shoots. It is necessary, however, that the spray should reach the under surfaces of the leaves, which is difficult to secure; nine-tenths of what falls on the upper surfaces are wasted. The gardens in the Darjeeling District are often on very steep slopes, and the transport of the material is an arduous task. The heavy rainfall too, at the time of year when the blight is spreading most rapidly, reduces the number of days suitable for spraying and often washes off the mixture before it has time to set. As the spray does not prevent young blisters from ripening, the coating should remain long enough on the leaves to check infection from subsequently matured spores, if it is to be effective. This is almost impossible to secure in the rains, and general spraying at this season is out of the question. But on heavy pruned tea, new extensions, and seed beds, where the area is usually small and the damage may be lasting, a sustained effort to check the disease by repeated sprayings has been found to repay the labour and expense. Further, cold-weather spraying, and also spraying in the early months of the year, when the first cases are seen, may be found of value. For cold-weather spraying, it has been recommended that badly infected tea should be first thoroughly cleaned out and then sprayed with a solution of 2 lb. caustic soda in 10 gallons water. This is said to have the advantage over Bordeaux mixture of not only killing fungi but also removing mosses and lichens; but it cannot be used on bushes with leaf as it burns the leaves. It is best applied some weeks before the new buds are due to appear. Where there is much leaf, as on unpruned, light pruned, and seed tea, Bordeaux mixture should be used, and applied about March. The cost of cold-weather spraying with Bordeaux mixture has been found to be from Rs. 2-4 to Rs. 2-8 per acre, excluding initial cost of machines only, and the labour required, 1½ men and 1 boy

for 4 hours. The strength used in the experiments on which this calculation is based was 6-6-100, and the water supply convenient.

The conditions which predispose the tea to attacks of this blight are not yet well known. It has been observed that the high quality "Assam" and "hybrid" varieties in Darjeeling are more severely attacked than the "China" and "Manipuri" kinds. In Assam also, the better varieties of "Assam indigenous" are said to have suffered severely in 1906, but the "hybrids" here were less damaged, while "Manipuri" in some places was almost immune, but in others was nearly as much affected as the "Assam."

Shade favours the blight, as also do low, moist localities. But the slight shade from properly planted and not too dense trees is said not to have any ill effects.

Unquestionably the really important external factor in the disease is the atmospheric humidity. In Assam, a heavy rainfall in February, March, and April, coupled as it usually is with a relatively low temperature, is intimately related with the outbreaks that have been observed. The immunity of much of the Assam tea areas is attributed, with great probability, to the fact that heavy rain in these months is rare outside the north-eastern corner in which the blight is prevalent. In Darjeeling, the moisture conditions are such as to favour the disease in many places: on many of the slopes, rain falls almost every day during the monsoon and mists hang about the hill sides. At elevations of from 4,000 to 5,500 feet, these conditions are most marked, and it is here that the worst damage has been done.

Though some half-dozen allied fungi have been found on the leaves of jungle trees and bushes in the Himalaya, that under consideration has not been found on any other plant but tea. The probability is that it lives all the year round on tea only, and that re-infection from the jungle, such as, no doubt, frequently occurs in red rust, need not be feared in blister blight.

**Pink disease** (*Corticium salmonicolor* B. & Br.).—This disease is known to attack tea in southern India, Ceylon, and Java, but has not yet been recorded in the northern tea districts of India. It is described below on p. 499.

**Brown root disease** (*Hymenochaete noxia* Berk.).—This is one of the diseases formerly included under the general name "stump rot," since it ordinarily arises, like the diseases caused by *Rosellinia* and

*Ustilina*, from the decaying stump of some jungle or shade tree, which has been cut down and left to rot in the soil in which the tea is growing. It is widely distributed in the tropics, occurring in Ceylon, the Malay Peninsula, Java, Sumatra, New Guinea, Samoa, and east and west Africa. It attacks a number of the most valuable tropical products and also jungle trees; rubber (*Hevea*, *Manihot*, *Castilloa*, *Funtumia*), coffee, cacao, camphor, breadfruit, Caravonica cotton, coca, dadap (*Erythrina* sp.), *Grevillea robusta*, *Cinnamomum Cassia*, *Albizia stipulata* and *Brunfelsia americana* being amongst its victims. It is the commonest root disease of *Hevea* rubber in Ceylon, and the only one known to attack cacao in that colony. In India, it has, so far, been noticed on tea, coffee, *Hevea*, *Manihot*, and *Grevillea* only, but will probably be found on other hosts. It is the commonest cause of root disease in tea in the sub-Himalayan tracts, especially in the sandy soils of the Duars and Terai. It is also found in Darjeeling.

The symptoms resemble those of the other root diseases mentioned below, but, as the spread is slow, it is usually observed when only one or a small group of bushes is affected. The leaves wither and the bush dies rather suddenly. On digging it up, the roots are found encrusted with a mass of sand, earth, and small stones, intermingled with bark and the mycelium of the parasite, the whole forming a gritty coating which is quite characteristic (Fig. 203). In the early stages the colour is brown, due partly to the tawny brown hyphæ of the fungus, but later on it turns black, almost as if charred. This is especially noticeable on the tap root. The brown sheets and masses of the fungus are found projecting from the outer surface, and also extending into the deeper layers of the cortex as far as the wood, where there may be a continuous fungal sheet, lying between the bark and the wood. The inner layers are light in colour, sometimes almost white. In the early stages, the wood of the root is little altered; later on, more or less wedge-shaped areas with a grey discoloration, bounded by clearly marked, narrow, sinuous, brownish lines are found, and if the bush be left for a long time after death, the tissues within these dark, boundary lines decay, leaving a honeycomb structure, the bounding marginal portions being dark and hard, while the intervening rotting portion is yellow and soft. The bark shreds easily into layers, each layer lined more or less by fungus; and flakes of it are found buried in the fungus mycelium and gritty coating on the root.

The mycelium of the parasite occurs in considerable quantity on the surface of affected roots. It consists of long, flattened hyphæ, varying in diameter, septate at rather wide

intervals, and branching sparingly. The hyphae are brown when young, and tend to collect into small sheets and nodules. Later on they usually condense into more continuous sheets, the outer layers of which are dark brown or black, hard, brittle, and rather shiny, and serve as a covering to the lighter coloured hyphae below. At points where extension is still going on, the hyphae can be found forcing their way between the cells of the cortex and down to the cambium layer. The wood is invaded at a later stage chiefly along the medullary rays, causing the wedge shaped areas mentioned above.

No mature fructification has as yet been detected on tea and there is no absolute proof that the fungus described is really parasitic, though this is highly probable from its regular occurrence. The sporophore of the presumed parasite *Hymenochaete noxia*, has been found outside India on several of the other hosts suffering from a similar disease including Hevea and breadfruit. It appears to be rarely formed so that dissemination by spores is probably uncommon. It develops as a thin dark brown crust adhering to the base of the stem and up to several inches in diameter. The surface is brittle apparently from the drying of a gummy substance excreted from the hyphae and is provided with numerous minute bristles each of which is a single rigid hypha up to  $130\mu$  long by 5 to  $8\mu$  broad, bright brown in colour with very thick walls and blunt or pointed at the tip. These bristles may sometimes be found even on sterile crusts of the fungus. When a hymenium is formed it covers a part or the whole of the exposed surface of the sporophore and is somewhat velvety in appearance from the presence of the bristles. The spores are formed freely at the ends of upright basidia and are elliptical and 7 by  $4\mu$  in diameter.



177 Brown root disease of tea (*Hymenochaete noxia*) immature fructification at base of tea stem.

In Ceylon, it is said to develop sometimes in old tea where no jungle stumps remain, but in one case was definitely traced to old nabor (*Mesua ferrea*) stumps. Dadap, Grevillea, Melia, Cassia, and Semecarpus can also start root disease. In India and in the Malay Peninsula, observations have led to the view that it always arises from decaying wood and that its propagation by spores must be very rare. If the dead bush or tree is allowed to remain, the mycelium spreads along the roots until it comes into contact with the roots of other bushes, which are then in turn attacked. It is unable to spread through the soil (unlike *Rosellinia arcuata*, for instance), and its extension is much slower than in the case of either *Rosellinia* or *Ustilina*. In one case observed in Hevea in Ceylon, the first death occurred (probably from infection from old cacao stumps) when the trees were 8 years old. It was not removed, and two years later the next tree in the line, 14 feet away, was killed. After another two years, the process was repeated. The path of the fungus from one tree to the next was traced along the roots. In India, the disease is most often met with on sandy soil.

The treatment is comparatively simple. Dead bushes, with as much of their roots as possible, should be removed and burnt. Where an old stump is found in the neighbourhood, it should also be dug out or broken up. If all infected roots are removed, it is rare to find any further cases, as the mycelium does not appear to be capable of independent life in the soil. Some have recommended that quicklime should be forked in, as it may help to destroy remnants of the fungus in roots left behind, but it is not certain that lime kills the fungi concerned in root disease. Trenching is unnecessary, if the roots are completely removed. There appears to be no reason why replanting may not be done at once, though further experience on this point would be advantageous. As in the case of other allied diseases, it would be most valuable to know what jungle and shade trees (or shrubs) are capable of starting this disease, so that, when it is necessary to cut them out, precautions may be taken to prevent their stumps from damaging the tea. *Mesua ferrea* stumps, at least 14 years old, have been known to kill tea in this manner in Ceylon, and probably several of the trees mentioned under stump rot kill tea by starting this form of root disease and not *Ustulina* or *Rosellinia*.

**Red root disease** (*Sphaerostilbe repens* B. & Br.).—This fungus has been recorded as a parasite of *Hevea* rubber trees in Ceylon and Malaya, and has been found on tea in Assam. It is not considered to be a serious enemy in either case; probably it requires special soil conditions to become parasitic at all on tea.

The fungus is confined to the underground parts of the plant, where it is visible on the base of the stem, and on the larger roots, as clusters of short, reddish stalks, of a maximum length of one-quarter of an inch and often with a round, white head (Fig. 178, 1). They are quite conspicuous when fresh. There is no visible external mycelium, such as occurs in the *Rosellinias*, but on lifting off the bark (which is usually decaying and easily removed), very characteristic, flattened, black rhizomorphs are exposed (Fig. 178, 8). These strands form branched markings on the surface of the wood, which persist after most of the fungus mycelium has decayed. The individual strands are about one-eighth of an inch across, and vary in colour, when fresh, from pink to brown or black, according to age; those that are quite young appear reddish, and in culture the reddish tinge is distinct.

The effect on rubber trees is said to be to thin the foliage and cause the branches gradually to die back. The progress of the disease is slow,

but death has been observed to result in both young and old trees. The effect on tea has not been fully recorded.

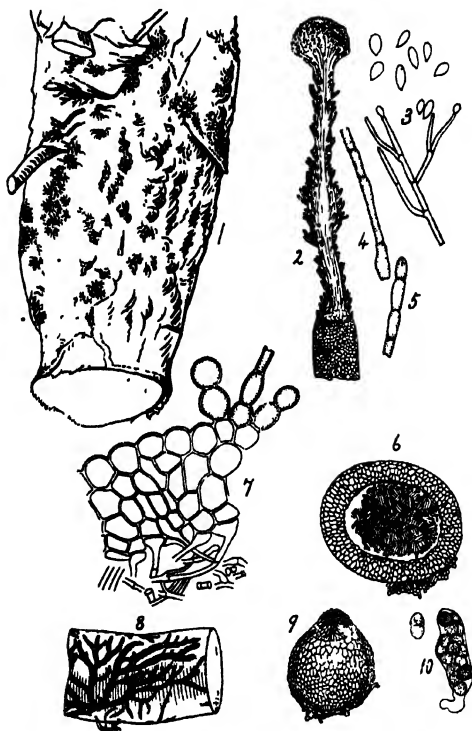


FIG. 178. Red root disease of tea (*Sphaerostilbs repens*). 1, root with conidial stage, nat. size; 2, compound sporophore,  $\times 33$ ; 3, fertile hyphae and conidia,  $\times 260$ ; 4, hypha with spiny wall from base of conidiophore,  $\times 260$ ; 5, hypha from side of conidiophore,  $\times 260$ ; 6, cross-section of rhizomorph showing cortex and medulla; 7, part of same showing proliferation of surface cells,  $\times 260$ ; 8, rhizomorph on surface of wood,  $\times 8$ ; 9, a perithecium; 10, ascus and ascospore,  $\times 260$ .

The mycelium extends throughout the cortex and wood of affected roots, and may advance some distance up the stem within the wood. The bark is softened and turned bluish-purple in the deeper layers. The wood also may be discoloured, usually becoming

bluish-black. Rhizomorphs are freely formed, chiefly between the bark and wood but also in the bark. They are also produced in culture, and consist of a cortical part, composed of polygonal pseudoparenchyma, and a medulla of thread-like hyphæ (Fig. 178, 6). The surface is often rough or even hairy, from the growth of the surface cells into chains of rounded cells or elongated hairs (Fig. 178, 7). Conidia of two kinds are formed in culture, both borne on the ordinary hyphæ of the mycelium and not on special sporophores. One kind is round, brown, thick-walled, and 10 to 12 $\mu$  in diameter, the other oval, colourless, thin-walled, and up to 20 $\mu$  long by 10 $\mu$  broad.

On the host plant, the conidial stage is represented by the prominent sporophores mentioned above as forming roddish tufts on the bark. Each stalk consists of a somewhat swollen base of pseudoparenchymatous cells, from which a cylindrical body of elongated hyphæ, closely united into a solid mass, arises (Fig. 178, 2). At the top, the hyphæ branch frequently and each branch terminates in a blunt tip on which the conidia are borne singly (Fig. 178, 3). After falling, they remain collected into a mass, embedded in a mucilaginous substance, on the end of the stalk. The cells of the stalk are roddish-brown in colour and give off laterally hair-like processes, consisting of a chain of cells, with roughened or spiny walls, each cell being about 20 to 26 by 4.5 to 9 $\mu$  in diameter (Fig. 178, 5). Hyphæ with similar walls occur at the base of the sporophore and on the surface of the rhizomorphs (Fig. 178, 4).

The conidia are oval, pointed at one end, hyaline, and 11 to 13 by 4 to 5.5 $\mu$  in diameter. Larger conidia, up to 22 by 10 $\mu$ , have been described on other plants, and the spores are probably very variable in size.

At a later stage, perithecia are formed on the bark. They are small, dark red bodies, rounded below and drawn out above into a short conical neck, at the tip of which is an opening (Fig. 178, 9). They are found scattered on the denser parts of the mycelium, or clustered around the base of the conidiophores, or even seated on the latter. On rubber, they are described as being about 600 $\mu$  high and 400 $\mu$  broad. On tea, those hitherto seen have been less than half this size. Each contains numerous aeci without paraphyses (Fig. 178, 10). The aeci are irregularly cylindrical, with a short stalk, and measure 75 to 200 by 10 to 20 $\mu$ . They contain 8 spores, in one or two rows. Each spore is composed of 2 cells, constricted at the septum, oval, pale reddish-brown, and 19 to 21 by 8 to 10 $\mu$  in diameter.

The parasitism of the fungus has not been established, inoculations on *Hevea* trees in Malaya having failed. If a true parasite, there would seem to be some special conditions requiring to be satisfied before infection can occur. Deficient drainage, and swampy, stiff, or sour soil, have been suggested as predisposing causes, but little is really known of the disease as yet.

The cases are usually scattered, but the fungus is capable of spread from one bush to another through the soil, as in stump rot due to *Rosellinia*, and it is said usually to originate in old jungle stumps.

Treatment, on present knowledge, should be on the same lines as in stump rot. The fungus lives readily as a saprophyte on pieces of fallen wood of several trees, and no stumps or old wood should be left near by. Trenching should be done both around the patch and between the rows, and slaked lime should be mixed with the soil at the rate of four lb. to every square yard.



Besides tea and rubber, the fungus has been found on jak (*Artocarpus integrifolia*), dadap (*Erythrina*), and arrowroot (*Marantia arundinacea*) in Ceylon, being undoubtedly parasitic on the latter. An allied disease is known on limes in Dominica, but the cause has not yet been identified definitely.

**Stump rot** (*Rosellinia* spp. and *Ustulina zonata* (Lév.) Sacc.).—The name "stump rot" has been somewhat indefinitely given to a group of diseases of tea, coffee, and other plants, which, while differing more or less in their symptoms, all agree in that the infection usually arises underground from the rotting stump of some jungle or shade tree, which has been cut down and left to decay in the midst of the tea, or when the land is first cleared for planting.

The term may conveniently be restricted to the forms of disease caused by parasites belonging to the allied genera, *Rosellinia* and *Ustulina*. The somewhat similar disease caused by *Hymenochaete noxia* has been separately considered under the name of "brown root disease," as the fungus concerned is a widely different one, and the characters sufficiently distinctive to have given this name a definite significance in other countries. So far, only these three genera have been identified in India, though it is probable that they do not cover all forms of stump rot which occur in this country. In particular, the type of disease caused by *Poria hypolateritia* in Ceylon may be expected to exist in India.

In stump rot caused by *Rosellinia* or *Ustulina*, the tea, especially in land cleared from heavy jungle, is found to die out in patches, without apparent cause. At first, a bush or several near together appear as if suffering from drought or insufficient nourishment. The leaves wither, turn brown, and drop off. In some cases, death is very sudden, almost as if from fire, and the leaves remain hanging and wilted, but still more or less green, on the bush. New shoots rarely arise from below, once wilting sets in, and this sometimes helps in distinguishing stump rot from forms of stem disease. Usually, after the first bushes die, others, surrounding them, show signs of infection, and the disease spreads in ever-widening circles unless measures to check it are taken. In most cases that have been examined, the decaying stump of some tree, formerly cut or blown down, has been found in the centre of the patch, and it is now fully accepted that the disease commonly arises from these stumps in some manner not yet entirely understood. This is especially the case with *Ustulina*, some of the Indian *Rosellinias* being much less often connected with stumps.

Stump rot occurs in all the tea districts of India. In the Terai, the form caused by *Ustulina* is, so far, the only type observed, and this also is the commonest in the Brahmaputra valley, where, however, that due to *Rosellinia* sometimes occurs, especially in old tea. In Sylhet, the latter is said to predominate. These reports are, however, liable to be modified as time goes on, as the different forms are not yet sufficiently known, particularly as regards *Rosellinia*, to make identification easy.

The same difficulty arises if we desire to ascertain the distribution of the different varieties of stump rot in other countries. The disease is well known in many tropical and sub-tropical lands, where tea, coffee, cacao, rubber, and other woody plants are cultivated. In several cases it has been attributed to *Rosellinias*, but the only species identified, outside India and Ceylon, are *R. bunodes* (B. & Br.) Sacc., and *R. Pepo* Pat., from the West Indies, where they attack limes in recently cleared forest land in Dominica, Castilloa and camphor in Grenada, and many other plants, including all that are usually planted in new forest clearings, in various places; while a similar disease in coffee and *Inga laurina* in Guadeloupe may very possibly be also caused by them. The black root fungus disease of coffee in Java may similarly be due to a *Rosellinia*; and in Singapore Botanic Gardens a form, named as *R. echinata* Mass., but indistinguishable from *R. bunodes*, was described some years ago as attacking *Ficus dubia* and a number of other trees and shrubs, and killing them rapidly.

In only a comparatively small number of cases has the parasite been identified in India. In most it has proved to be *Ustulina zonata*, but *Rosellinia arcuata* occurs in Darjeeling, another species of this genus in Dehra Dun, and, in Assam, *Rosellinia* is said to be a cause of stump rot in old tea in certain districts, though the species, as in the Dehra Dun cases, has not yet been identified. In Ceylon, *Ustulina zonata* is the most frequent root parasite of tea, but *Rosellinia arcuata* also does harm. In South India, *R. bunodes* kills pepper and several forest and shade trees, but has not yet been found on tea. The parasitism of this species, first detected in India, seems to have been doubted in Ceylon, but the recent West Indian investigations support our views as to its danger. It is more fully described above on p. 357. It is evident from all this, that several allied species are capable of attacking tea; and it is by no means certain yet in what districts they occur and which is the commonest cause of stump rot in each area.

The *Rosellinia* found in a typical case of stump rot in Dehra Dun, only developed its perithecial form, after some months, on the rotting main roots. This showed it to

be one of the rare forms in which the perithecia develop in definite sclerotia, as in the well-known vine parasite, *Rosellinia necatrix*, in Europe. Further identification was impossible, and the case is only of interest as being one of the few so far recorded of the discovery of the perfect stage of *Rosellinia* on tea in India.

*Rosellinia arcuata* Petch has quite recently been identified as a parasite of tea in northern India but has been chiefly studied in Ceylon. The species is interesting, as it is believed to originate usually in accumulations of dead leaves and not, like most other soil-dwelling parasitic members of the genus, in old wood, though it has been observed in some cases to start from *Grevillea* or *Symplocos*. The mycelium is found in the top two or three inches of the soil, especially where there is a mulch of dead leaves, and spreads very rapidly in the form of somewhat fleecy strands, attacking the roots of several other plants besides tea. The growing margin of the mycelium is white, further back it is smoky-grey, and when old quite black. The hyphæ spread down the main roots when they reach a tea bush, forming a loose, cobwebby mass on the surface or, especially in old bushes, black strands, closely adherent to the root. These enter the bark and spread out into star-like sheets of white mycelium, up to half an inch in diameter and easily seen on peeling the bark from the large roots (Figs. 179, 9). The attack usually begins at the collar and while some hyphæ extend down the roots others grow upwards and form a coating, purple-grey at first and then black, on the stem for about 6 inches above



FIG 179. *Rosellinia arcuata*. 1, perithecial stage,  $\times \frac{1}{2}$ ; 2, group of perithecia,  $\times \frac{1}{2}$ ; 3, conidial stage,  $\times \frac{1}{2}$ ; 4, asci and paraphyses,  $\times 100$ ; 5, ascospores,  $\times 210$ ; 6, young compound conidiophore,  $\times 100$ ; 7, apex of fertile hypha of same,  $\times 210$ ; 8, ditto of older conidiophore,  $\times 210$ ; 9, mycelial strands under bark of root,  $\times \frac{1}{2}$ .

ground. Here they develop a conidial stage. The perithecial stage appears late and, as with most of its allies, is rarely found in the field. It has been suggested that, owing to the free formation of conidia, this species is capable of dissemination by the wind and that the ordinary measures to prevent the spread of the mycelium in the soil, used in dealing with stump rot, are ineffective unless supplemented by arrangements to check dissemination of the conidia. The fungus attacks *Grevillea robusta*, *Symplocos obtusa*, *Panax fruticosum*, camphor, *Strobilanthes*, and *Erythrina*, in Ceylon, but not cacao or Hevea.

The conidial stage of *R. arcuata* (Fig. 179, 3) consists of short, erect, bristle-like stalks, produced in dense clusters, so as to give a velvety appearance to the surface. These stalks are branched above (Fig. 179, 6), each branch bearing hyaline, unicellular, narrow-oval conidia, 4 to 6 by  $2\mu$  in diameter (Fig. 179, 7-8), which form a white or greyish powder on the surface. The perithecia are spherical, black bodies, almost like grains of shot, about  $\frac{1}{16}$  inch in diameter, and found in clusters on the mycelium at the base of the stem, often amongst the conidial stalks, so as to appear at first half-immersed in the mycelium (Fig. 179, 1-2). The asci are 280 to 350 by 7 to  $9\mu$ , cylindrical, with a single row of spores, 8 in number (Fig. 179, 4). Paraphyses are intermingled with the asci and are thread-like bodies, about  $2\mu$  across. The spores are opaque black, boat-shaped, with pointed ends, and measure 40 to 47 by 5 to  $7\mu$  (Fig. 179, 5).

*Ustulina zonata* (Lév.) Sacc. is perhaps the commonest cause of

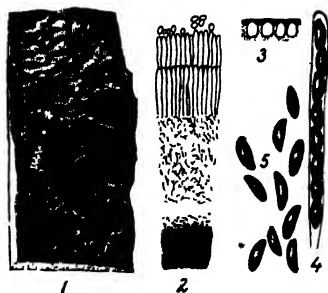


FIG. 180. *Ustulina zonata*: 1, fructification at base of tea stem,  $\times 1$ ; 2, section of upper part of conidial fructification,  $\times 315$ ; 3, perithecial layer at surface of mature fructification,  $\times 2$ ; 4, ascus,  $\times 180$ ; 5, ascospores,  $\times 200$ . (8 after Brooks).

stump rot both in India and Ceylon, and has recently attracted much attention on rubber estates in Malaya. It is often found in the Terai, Nowgong, Dibrugarh, Doom-Dooma, and other parts of the north-eastern tea districts but has not yet been reported from southern India. Like the brown root disease, it is said to be found usually in recently cleared land, whereas *Rosellinia* is commonest in old tea. It arises frequently from decaying stumps of *Grevillea*

and *Albizzia moluccana*, in Ceylon, where both are much used as shade trees in tea gardens. Such decaying stumps are said to be subject to infection by means of the spores of the fungus, while the tea is usually attacked only below ground by the mycelium from the shade

tree stumps. When first noticed, the stump is usually in the last stages of decay and two or three of the surrounding tea bushes are dead. As a rule, the bushes die gradually, the leaves fall off and no new shoots are produced; sometimes, however, they dry up suddenly, with all their leaves attached and still greenish.

The dead tea roots do not show any external mycelium, unlike what occurs in *Rosellinia arcuata* and probably most other species of the latter genus, where a surface mycelium is usually well developed. Only a few inconspicuous black spots mark the places where the internal mycelium is emerging to form fructifications. These fail to progress further unless above the soil level. On removing the cortex of the root, white, fan-shaped patches of mycelium are found, overlying the wood; these are frequently fused into a continuous sheet. Irregular, black lines are seen in a cross-section of the root.

The fructifications are produced abundantly on the dead *Grevillea* stumps, and also at the collar of the tea stem, if left standing long enough (Fig. 180, 1). The mycelium spreads out on the surface, forming a yellowish-white plate, lying close to the bark but only attached at one point, the rest of the under surface being at first white and later black. The plate is up to 2 inches or more in diameter, by about  $\frac{1}{4}$  inch thick, and is concentrically furrowed owing to alternate periods of growth and rest. Frequently, several such plates coalesce to form a larger sheet, and irregular crusts up to a foot or more across may thus be formed.

When young and still soft, conidia arise from the surface (Fig. 180, 2). These are hyaline, unicellular, narrow-oval, and measure 4 to 8 by 2 to 3 $\mu$ . They arise on closely crowded, erect, simple stalks, and give a grey or greenish, powdery appearance to the surface. Later on the plate becomes purple-grey (sometimes black when old), hard, and brittle and the surface is dotted with small spots, the openings of the perithecia. The perithecia are round or oblong, about 1 mm. in diameter, and entirely buried in the plate, the mouths scarcely projecting (Fig. 180, 3). The asci are cylindrical, about 250 by 10 $\mu$ , with long stalks, and contain each 8 spores (Fig. 180, 4). Thread-like paraphyses occur between them. The spores are opaque black-brown, boat-shaped, unseptate, and with obtuse ends (Fig. 180, 5). They measure 27 to 38 by 7 to 13 $\mu$ .

The above description applies only to typical forms, the fungus being very variable as regards the size and shape of the fructification. The plates may be undulating or thrown into prominent folds, especially when found on loose bark and earth at the base of a *Grevillea* stump. Sometimes they are stalked, and there may be a number of such stalks each with a small plate at the top, the appearance in the conidial stage resembling a small *Xylaria*. In sheltered places, the upper surface may remain white, even when bearing perithecia.

*Ustulina* may usually be distinguished from *Rosellinia* by the absence of any external mycelium, the characteristic sporophores, when present, and the fan-shaped, white mycelial sheets under the bark, those of *Rosellinia* being more star-shaped. The different species of *Rosellinia* can only be distinguished accurately in the perithecial stage, which is late in appearing and often hard to find.

The fungus also attacks the pomelo (*Citrus decumana*), *Grevillea robusta*, *Albizzia moluccana*, *Cassia nodosa*, and *Lafansia Vandelliana*, besides probably other jungle trees. Its spores germinate readily on dead wood, and it has been found, amongst other hosts, on dead coconut stem, but whether parasitic or not is not certain. It occurs in Ceylon on old, dead Hevea wood, and does much damage to full-grown rubber trees in Malaya. On Hevea it is a wound parasite, following borer attacks, bark injuries, and broken branches; contact infection from diseased roots below ground also occurs. It sets up a dry rot of the wood of the stem and roots, being especially frequent at the collar. In late stages, the affected wood can be crumbled to shreds between the fingers. The attack is often unilateral, extension occurs chiefly in the wood, and the death of the tree may be rapid or very gradual. Trees under 10 years old are seldom affected. Thus, on this host, the fungus is a direct parasite, and jungle stumps and old timber are not concerned, except in so far as that they maintain a constant supply of infective material. Tea, on the other hand, appears not to be often attacked directly, but only through the agency of a neighbouring stump or another infected plant, and usually only underground. Exceptionally, tea may become infected through dead snags above ground. It seems to pass readily from root to root, but has not been proved to travel through the soil, nor to form mycelial strands in the soil like *Rosellinia*.

The parasite is known in Java, Malaya, and possibly South America, but seems to have been sometimes mistaken for *Ustilina vulgaris*, a common European species.

The treatment of stump rot, from whatever cause, must be, in the present state of our knowledge, uniformly directed to preventing spread of the disease, through the soil or along the roots, from bush to bush. The danger of aerial spread from spores produced above the ground level is probably slight, if early precautions are taken to remove the dead and visibly infected bushes, and not to leave stumps of *Grevillea* and other susceptible trees projecting above ground or lying on it.

When the first case is detected, in the neighbourhood of a rotting stump, a trench should be made, to include not only such bushes as show obvious signs of attack but also at least one row further out. The trench should be about 18 inches deep, and may be as narrow as consistent with convenience in digging. In the heavy Malayan jungle lands in which rubber has been so extensively planted of recent years, it is recommended to trench to 2½ feet, in order to get below the level of infected lateral

roots of jungle trees, where stump rot is prevalent. In this case, the fungus *Fomes lignosus* (*semitostus*) is the usual cause of the disease, and it is doubtful whether *Rosellinia* or *Ustulina* ever go much deeper than a foot. The soil from the trench should be thrown into the infected area, and care should, of course, be taken to keep the channel free from débris, particularly leaves and twigs, by periodical cleaning out.

The diseased bushes should now be carefully removed, with as much of their root system as can be got out, and the whole burnt on the spot, without delay. It will often be possible, during this operation, to examine the roots of the other bushes, within the trench, especially as there are usually indications of the side from which disease has come and the direction in which it is spreading to the next bush. If the roots of the bushes are infected, they should be removed also and burnt. If not, they may be left and kept under observation for a time. The top soil should be well turned over, and if the rotting stump from which the disease has originated can be located, it should be broken up and burnt if possible. Roots and other parts which are difficult to burn, owing to their dampness, may be scorched sufficiently to kill the surface layers of fungus by mixing with straw or the like, and can afterwards be burnt at leisure. If the bushes are left long before removal, it is advisable to scorch before uprooting, as there may be a development of spores at the base of the stem, and, consequently, danger of disseminating the fungus during the operations. Some recommend a second digging over, and the incorporation of lime at the rate of about 25 lb. to every 100 square feet of surface, after about a month, and there is no doubt of the advantage of keeping the soil stirred and well aerated, though the benefit of liming seems to require more detailed proof, as it is known to be useless against several soil-dwelling parasites. A more elaborate method has recently been advocated in Java, and has been tried in India. It aims at the production of ammonia gas in the soil, though here again it must be said that it has not been proved that ammonia interferes with the activity of the specific fungi under consideration. The operation consists in first liming the soil and then adding ammonium sulphate, the reaction between these substances producing ammonia. It has not replaced the older methods. *Rosellinia arcuata* can often be checked by clearing away leaves and twigs, cutting out infected parts and applying a good fungicide to the diseased bushes, exposing the collar of the bushes near by, and forking in 2 lb. lime per square yard.

It seems probable, though exact observations on this point are lacking, that the parasitic mycelium rapidly disappears from soil that

has been completely cleared of rotting wood, diseased roots, and other plant debris. It is said in Ceylon to be waste of time to try to replant in patches caused by *Rosellinia arcuata* before 18 months. In South India, replanting after a year has been successful, but it is possible that the bushes had died from brown root disease, in which it is known that supplies may be put in very early if all the infected roots are removed. On present knowledge, it is perhaps safer not to replant for 18 months.

There is no accurate information as to the conditions which are favourable to stump rot. *Ustilina* has been frequently observed on sandy soil. *Rosellinia* is said, on the other hand, to be worse on low-lying, badly drained, and sour or heavy soils. It is also said to be worse in heavily shaded localities. Further knowledge of its dependence on such external conditions is desirable.

The trees whose stumps are capable of starting tea root diseases in India are known in a few cases, but not what kind of disease each can set up. The worst are said to be the madar (*Erythrina indica*) and bela (? *Semecarpus Anacardium*). Others are the sum (*Machilus bombycina*), bukhain (*Melia Azedarach*), simul (*Bombax malabaricum*), *Grevillea robusta*, and to a less extent nahor (*Mesua ferrea*). The sau (*Albizia stipulata*), so much used as a shade tree, is said to be, perhaps, the least dangerous of all. These observations apply to Assam only. In South India, the trees likely to set up root disease have been noted chiefly in connection with coffee, and are referred to under that head (p. 480). *Grevillea*, *Symplocos*, and *Lagerstræmia lanceolata* (benteak) are certain offenders. In Ceylon, *Rosellinia arcuata* may start from *Grevillea* and *Symplocos*, and *Ustilina* from *Grevillea*, *Albizia moluccana*, and the other hosts mentioned above as subject to attack by this fungus.

The question whether it is financially profitable to clear out stumps, when opening up jungle lands, seems to depend on the special circumstances of each case. The arguments in favour of it are decidedly less strong than in the case of rubber (see p. 508), the latter being much more subject to root disease, and more troublesome to replant when vacancies occur. It is clear that certain trees, such as the simul, should not be cut down at all, unless their stumps can be removed when clearing. The choice of *Grevillea* as a shade tree seems to have been unfortunate, as this tree also cannot safely be cut down without "stumping." It can sometimes be pollarded, without dying, when it gets too big. If a complete list of the dangerous trees were available, it would probably often be possible to carry out a selective stumping, removing only such



stumps as are known to do harm. In Malayan rubber estates, selective stumping is said to be impracticable, but the number of harmful trees is greatly superior in that region, owing to the prevalence of the *Fomes* root fungus on a very large number of hosts. In any case it appears foolish to leave the stumps of such trees as are mentioned above as being known to cause trouble, and these should either be stumped or not felled.

**Copper blight** (*Laestadia Camelliae* Cke. = ? *L. Theae* Rac.).—This disease is prevalent in certain tea districts and has attracted some notice during the last few years, owing to the extreme rapidity with which it spreads, though, on the whole, it is not considered to do much damage.

It was first described from Johore in 1884, the two stages of the parasite being taken for distinct fungi and named *Sphaerella* (*Laestadia*) *Camelliae* and *Phoma Camelliae* respectively. *Laestadia Theae* Rac. was subsequently described from Java, on leaves bearing *Colletotrichum Camelliae*, and was taken to be probably the perfect stage of the brown blight fungus. If this is so, the Java fungus is identical with *Glomerella cingulata* and has nothing to do with copper blight, but as the two fungi are very similar in their microscopic characters, it is not certain from the description whether *Laestadia Camelliae* or *Glomerella cingulata* was observed. More recently, copper blight has been reported from Ceylon and the Federated Malay States. In India, it is known in the Duars and Terai, the Assam Valley, and Sylhet.

The first symptom is the appearance of a small yellowish-brown spot, soon turning copper-coloured, on the upper surface of the leaf.

This spot is irregular in shape and the margin not very clearly defined, merging into the green part of the leaf. The surface of the discoloured patch is covered with very minute,

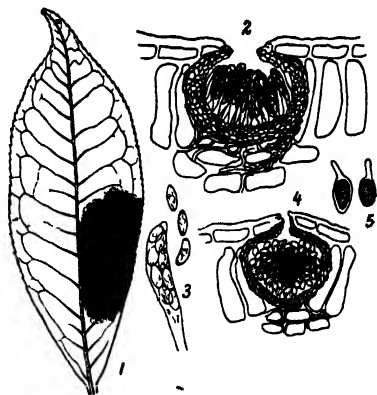


FIG. 181. Copper blight of tea (*Laestadia Camelliae*): 1, leaf with patch of copper blight,  $\times \frac{1}{2}$ ; 2, a perithecium,  $\times 200$ ; 3, ascus and ascospores of *Laestadia Theae*,  $\times 200$ ; 4, pyrenidium,  $\times 200$ ; 5, pyrenospores,  $\times 200$ . (3 after Bernard).

black dots, which on microscopic examination prove to be areas in which the peripheral leaf tissues have disintegrated, leaving small, crater-like depressions. In later stages the discoloration extends right through the leaf, appearing as a yellowish-brown patch on the under surface, the colour on the upper surface at the same time turning greyish, and a sharp margin appearing between the healthy and diseased leaf areas. Fully developed patches may be two inches or more in diameter. The diseased portion is very brittle and often traversed by numerous cracks.

In the early stages, especially at certain times of the year, the parasite, after extending a comparatively short distance through the leaf tissues, produces a pycnidial type of fructification (*Phoma Camelliae* Cke.). This consists of rounded, minute spore-cases entirely sunken in the tissues, scattered, and up to about  $100\mu$  in diameter (Fig. 181, 4). The mouth penetrates the leaf epidermis to open at the surface as a very tiny orifice. Later, when fully ripe, the whole upper part of the pycnidium, together with the leaf tissue overlying it, is thrown off, leaving a crater-like cavity. The inside is filled with small, oval, hyaline, unseptate spores, measuring about 10 by  $5\mu$ . Each is surrounded by a mucilaginous coat and has a tail of the same substance at one end (Fig. 181, 5). This spore form is chiefly found in the cold weather.

In the warmer months, especially when the patches turn grey above, the perfect type of reproduction develops as similar but larger black dots. These are the perithecia and contain ascospores (Fig. 181, 2). The perithecia are up to about  $250\mu$  in diameter, and are sometimes formed in concentric rings. They contain clavate aeci, about 50 to 60 by 10 to  $12\mu$  in diameter, without paraphyses, and each with eight spores. The spores are colourless, elliptical, unseptate, about 12 by  $5\mu$  in diameter, and arranged in two rows in the ascus.

The effect on the leaves is to dry them up, and cause them to become brittle and crack. The older leaves are chiefly affected. Sometimes there is distortion and folding of the leaves, when younger ones are attacked. The spores have not been germinated, and it is not known how infection occurs, but very probably the chief spread is from the pycnidial type of spore, the other being less easily set free. Spread in suitable climatic conditions is extraordinarily rapid, but ceases when a change of weather sets in. Shade has also a marked effect, the attack being sometimes confined to the shaded part of a bush. In Assam, it is said to develop most when warm sunshine follows heavy rain, as often occurs in April. The well-known grape disease ("black rot"), due to an allied fungus (*Lasdadia* or *Guignardia Bidwellii*), is equally dependent on the weather conditions prevalent at certain times of the year. It usually appears when, after a fall of temperature, accompanied by rain, the temperature again rises. It seems to be at this moment only that infection occurs. Some observers state that a light preliminary rain, a day or two before the heavy rain and fall of temperature, is also necessary.

Though widespread, copper blight is said to be not severe enough to necessitate any very drastic measures to check it. Where prevalent, it is certainly wise to remove the diseased leaves during the cold weather and, especially, to prune carefully any severely attacked bushes. If spraying is considered advisable, the exact time to do it must be worked out by closely watching the onset of the climatic conditions which induce an attack. The allied black rot of the grape is notoriously difficult to spray at the right moment, and treatment at other periods is of little use. Bordeaux mixture is the best to use.

**Internal root disease** (*Thyridaria tarda* Bancr. = *Botryodiplodia Theobromæ* Pat.).—The general characters of this parasite, and its effects on sugarcane, have been described above (p. 385), where it was shown that sugarcane stems are, as a rule, only attacked by it through wounds, or when they have been weakened by some other cause. On tea, however, it has hitherto been found chiefly in the roots, and there is no evidence that the infected bushes had been previously weakened in any way. It seems to be a true parasite on this host. Unlike most of the root diseases of tea already discussed, its attacks do not, in such cases as have been carefully examined in India, take their origin from decaying jungle or shade tree stumps; in Ceylon it is said to arise sometimes around cut-back Albizzias, but even in this case the Albizzias appear to have been attacked on the cut surface above ground, and may merely have shed a sufficient supply of spores into the air to infect the soil around them. In Bishnath and Dibrugarh Districts, in Assam, it has been found chiefly on cleared grass land, where tree stumps are rare, and not on land cleared from forest jungle, where *Ustilina* and *Hymenochaete* are the chief causes of root disease. It has not yet been recorded on tea outside India and Ceylon; its distribution even in this country is not accurately known; but it probably occurs throughout the tea districts, and it is unlikely that so widespread a fungus should fail to attack tea in other parts of the world.

The worst outbreak of this disease hitherto reported in India, occurred in Bishnath District, Assam, in 1901. In one garden about 100 acres were affected, the loss of bushes varying, by 1902, from 75 per cent. in some parts, to about 15 per cent. in others, the average for the whole being about 30 per cent. The affected area was originally grass land, not forest. Other subsidiary attacks were observed about the same time, the tea being in every case grown from the same seed, obtained from Dibrugarh District, where, as already stated, the disease is known to

occur. The bushes were from 3 to 4 years old and the attack began after the first pruning, when about 3 years old. Certain batches of the seed (it is believed, certain boxes) invariably gave rise to infected plants, while others remained healthy, and the planters concerned were convinced that the seed was already diseased when sown, the field evidence, on the whole, supporting this view. At the same time, there is reason to believe from other cases observed, that infection frequently occurs not through the seed but from the soil, or through wound infection above ground.

The symptoms are simple : the leaves take on a yellow appearance,

and the network of veins in the leaf becomes very prominent ; defoliation then gradually occurs, and branch by branch the bush dies. To the naked eye there is little alteration in the roots, especially the larger ones, those of intermediate size may show peculiar greyish markings, or rather large grey patches on the bark. On peeling off the bark, the wood of the affected roots appears stained by streaky, blue-black markings. There is no visible external mycelium, and only with a lens can the fructifications of the fungus be



FIG. 182. Internal root disease of tea (*Botryodiplodia Theobromae*): section of root showing effect on wood, nat. size; pycnidia in bark of large root,  $\times 2$ ; and, below, a pycnidium bursting through the bark, more highly magnified.

discerned, breaking through the bark of roots killed by the disease, as small, black pustules. The bushes usually, in the cases noticed in India, die out singly, not in ever-widening patches as in stump rot or brown root disease.

Besides these cases in young tea, others have been observed in old bushes in various districts. They were not easily distinguished from stump rot, the withered leaves remaining hanging on the bush for some

time. In many of these cases, infection clearly occurred through the wounds left after pruning.

In Ceylon, this parasite is said to kill the bushes most usually after pruning, and to cause a larger number of bushes to die in each individual attack than in any other root disease, though less common as a whole than *Ustilina*. Patches of from 40 to 100 dead bushes are not uncommon in the low country of the Island, and in one case more than one-tenth of the bushes were killed over an area of 15 acres. Losses of 25 or even 50 per cent. are said to occur in some blocks. Death usually takes place from six weeks to three months after pruning. This death after pruning is in harmony with what is known of the parasitic characters of the fungus on other hosts, as pruning necessarily causes some degree of shock to the bushes ; at the same time, there is good evidence that it can attack quite sound, strong bushes, and there is reason to believe that the fungus is already in the roots before pruning, the latter operation merely hastening its activity.

The mycelium is wholly internal, occupying the cortex and wood, which are blackened from the presence of the dark brown hyphæ. The fructifications are less evident than on sugarcane, the pyrenidia being buried, except the mouth. The microscopic characters agree with those described on sugarcane (Fig. 161). It is probable that the attack begins on the smaller roots, when infection proceeds from the soil, and works back to the main roots and along the central wood of these. As the inner wood is usually not functional, little apparent harm is caused until spread to the outer layers occurs. It has been suggested that, after pruning, the outer layers become partially dried and that this may allow the fungus to invade them more readily and explains the death of recently pruned bushes. Drought seems sometimes to act in the same way.

The disease is often found in India on sandy soil liable to drought. Many of the cases in old tea indicate that infection occurs through wounds, such as those caused in pruning, and that extension down to the roots subsequently takes place. The Bishnath experience, on the other hand, suggested seed contamination. We know, from other sources, that the fungus is commonly present in cultivated soil, as for instance at Pusa, but is usually able to attack plants such as indigo, growing in infected soil, only when they have been weakened from some other cause. In Ceylon, it is believed that buried tea prunings lead to an accumulation of the fungus in the soil, and that as the tea roots grow towards the rotting prunings, they become infected. In St. Lucia, in the West Indies, cacao is attacked at the roots in heavy, low-lying, ill-drained soils, and the fungus is said to be probably able to attack only under such conditions.

The progress of the disease on old bushes is slow. Young plants seem to be killed more quickly, but as the parasite may be present for

a considerable time before causing symptoms, it is difficult to gauge the duration of an attack.

The treatment consists in uprooting and burning affected bushes in order to check spore production, forking in quicklime, with a view to aid the destruction of plant débris which may harbour the fungus, and reducing the amount of prunings and other woody material that reaches the soil. Supplies have been safely put in very soon after removing the bushes. An application of a 2 per cent. solution of ferrous sulphate was tried round the base of the bushes in Assam, and at first believed to have given good results, but later reports are wanting. Good drainage is said in the West Indies to be most important. It is unnecessary to emphasise the advantage of planting from good seed only.

**Brown blight** (*Glomerella cingulata* (Stonem.) S. & v. S. = *Colletotrichum Camelliae* Mass.).—This disease was first described from Ceylon specimens in 1899, and was soon after recognised as being prevalent in India. It has since been reported from Java, Formosa, Uganda, and the Caucasus, and is doubtless widespread. A fungus found on dead, brown areas of the tea leaf in Alabama, United States, is, from the description, scarcely distinct, but has been named *Colletotrichum Carver*

Ell. & Ever. The perfect (*Glomerella*) stage, has only been found on greenhouse plants in Washington, United States, but there is said to be no doubt that the Indian form is identical.

When first noticed in Ceylon, brown blight seems to have caused some alarm. Little has, however, been heard of it of late. In South India, it has recently become prevalent in certain districts, and in most of the tea districts it is sometimes a cause of loss. In Ceylon and Uganda, it is

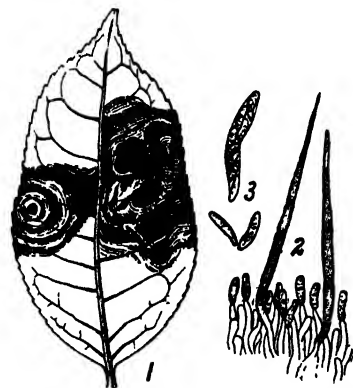


FIG. 183. Brown blight of tea (*Glomerella cingulata*); 1, leaf with brown blight, nat. size; 2, part of acervulus with conidia and setae,  $\times 280$ ; 3, ascus,  $\times 300$ , and ascospores,  $\times 400$ . (2 after Massée, 3 after Shear and Wood).

considered worse than grey blight, but in Java and the Caucasus, it has not been reported to be particularly harmful.

The first symptom is the appearance of small, yellowish-brown spots on the upper surface of the leaf, which extend both laterally and through the leaf, causing similar spots on the under surface. The colour soon deepens to chocolate-brown, and the growing margin, particularly on the upper surface, becomes marked by concentric zones of lighter and deeper shade, which are clearer than in any other leaf disease of tea. The margin of the leaf is frequently affected, and if growth has not finished in the leaf, the effect of the local death of the tissues is sometimes to cause bending or folding of the leaf at the diseased spot. There is a well-defined edge to the brown area, and outside this the cells are slightly yellowed, as can be well seen if the leaf is held up to the light. Scattered on the brown portions on both surfaces of the leaf, the acervuli of the fungus can be seen as tiny black dots, which are often somewhat hairy when seen with a lens. These are usually in concentric, curved lines towards the edge of the spot. When old, the central part of the diseased area often ruptures and falls out, leaving large holes in the leaf. The spots, when full-grown, may be up to an inch or more in diameter, and when several occur together they may coalesce to form large irregular patches.

The mycelium spreads within the leaf tissues, probably with alternate periods of growth and rest, and produces spores towards the centre of the spot while marginal growth is still going on. The acervuli arise from stromatic collections of the mycelium under the epidermis of both surfaces. They are up to about  $300\mu$  in diameter. From the surface, a layer of spore-bearing stalks grows out, rupturing the epidermis and exposing the spores to the outer air. The stalks are hyaline, up to about  $15\mu$  long by 3 to  $4\mu$  broad, and closely crowded together. The spores are borne singly at the tips, and are hyaline (pinkish in mass), unseptate, straight, rounded at the ends and 15 to 17 by 4 to  $5\mu$  in diameter. In many acervuli (but not in all) there are scattered, greenish-brown bristles (setae), with 1 or 2 septa, pointed above, and 100 to  $135\mu$  long by 7 to  $8\mu$  broad.

The Glomerella stage has not yet been found on tea in India, though what is probably the same occurs on chullies (p. 355). It will be more fully described under rubber (p. 512).

The disease is found more or less all the year round, but is most in evidence during the rains. It is usually confined to the leaves, though it has been reported on the stem of nursery plants in South India and, as will be mentioned below, an allied fungus is associated in Assam with a twig disease not yet fully investigated. Leaves of all ages are attacked, except the very youngest (those actually plucked). The effect of a bad attack in old tea is to weaken the wood, through defoliation, so that there is nothing to prune on to. If collar pruning is tried, the new suckers are apt to be attacked again. Supplies are also said to be much injured. It is said in Ceylon to spread with great rapidity, and also to do much damage to recently pruned tea, but it seems likely that it has

sometimes been confused with copper blight in that colony and these remarks possibly refer to the latter disease. No experiments have been made to test the mode of infection and other details in the life-history, and hence our knowledge of the parasite is lacking in many respects.

The following recommendations for treatment have been made. In nurseries, all attacked leaves should be picked off and destroyed as soon as the disease appears, and the plants should then be sprayed with Bordeaux mixture (5-4-50). A second spraying should be given if there is any sign of recurrence. All dead and dying plants should be removed and all fallen leaves collected and burnt. Slaked lime should be sprinkled on the surface of the ground and lightly worked in. The nurseries should be kept as open to light and air and as dry as is consistent with the well-being of the plants. Overcrowding and weeds should be avoided, and watering done only when necessary. Supplies and young clearings should be sprayed after removing attacked leaves, and any neighbouring older bushes also sprayed. In old tea, jungle and fallen leaves should be cleared up and slaked lime worked into the top soil at the rate of 5 to 10 cwt. per acre. Nitrogenous manures should be avoided, and those which help to produce hard wood, such as basic slag and sulphate of potash (or wood ashes), used. The bushes should be sprayed and the spraying repeated, if necessary, at one or two months' intervals. Three sprayings, in February, March, and May, have given good results in South India, when combined with regular destruction of diseased leaves and the application of lime and basic slag.

A disease which is sometimes known in the Assam valley as "H" blight, and is considered by more than one observer to have dangerous potentialities, appears to be caused by the attack of a fungus allied to *Colletotrichum Camelliae*, on the younger twigs. The result is a "die back" or "wither tip" disease, of the same general type as the coffee twig disease (p. 483). The attack occurs at or near the tip of the twigs and spreads backwards to the older wood. Fructifications of the fungus are found in the bark in abundance before the twig is dead. They occur as acervuli of the *Glucosporium* type (that is, similar to *Colletotrichum* but without the sterile bristles amongst the spores), and are smaller and with smaller spores than those of the brown blight fungus. As, however, the information available is scanty, it is premature to express an opinion as to the identity of the two forms. Many, perhaps most, of the allied parasitic species are capable of attacking both leaves and young twigs, and often do most damage in the latter position.



Another *Gloeosporium* has been found on the leaves of tea in German East Africa, and described under the name of *G. Theae* Zimm. It is very doubtful if it is distinct from *Colletotrichum Camelliae*.

**Grey blight** (*Pestalozzia Theae* Sawada).—This is said to be the commonest leaf blight of tea in India. It was known in Assam for many years before the first scientific description of it was published in 1898. Its occurrence in Ceylon was signalled in the following year, and it has also been reported from Java, the Caucasus, and Formosa. The parasite was believed for several years to be identical with *P. Guepini* Desm., a fungus commonly found on flowering forms of *Camellia* in many parts of the world, but of recent years has been identified in Java and Ceylon as *P. palmarum*, a widely-distributed parasite of the coconut. It differs, however, from both these fungi in important particulars.\* In India, it is said to occur in all the tea districts.

The attack begins on the upper surface of the older and harder leaves as minute, brownish spots which soon turn grey. These increase in size and coalesce, forming patches of variable extent and irregular outline, which sometimes involve the greater part of the leaf, and are especially conspicuous near the margins. The grey colour extends over the whole surface of the patch except at the growing edge, and the older parts may turn almost white. The margin is deep brown, forming a ring of variable breadth, from  $\frac{1}{16}$  up to  $\frac{1}{4}$  inch in diameter, round the central grey part. Outside this there is often a translucent band, shading into the green, healthy part of the leaf. The spot early appears on the under surface also, but remains usually light brown on this side, rarely turning to grey. On the upper surface, there may often be seen a series of narrow, concentric rings of alternate lighter and deeper shade, especially near the margin; occasionally these may be found underneath also but often they are absent from both surfaces. The fructifications of the parasite appear chiefly on the upper surface, a few only being sometimes found below. They are usually most numerous towards the margin, where they are often arranged in curved, concentric lines; sometimes they are scattered without order over the whole spot. Their numbers vary greatly, probably in accordance with the humidity or

---

\* Specimens on tea from India, Ceylon, and Formosa were compared by the author at Kew, in 1914, with the type specimen of *P. palmarum*, and with Desmazières' specimens of *P. Guepini* from the type locality (Angers). The three fungi were found to be distinct. Subsequently (1915) Sawada published his diagnosis of *P. Theae*, and specimens kindly communicated by him are identical with the Indian and Ceylon material.

other climatic conditions which prevail during the growth of the spot ; in some well-developed spots they are scarce or even absent. They vary from about  $\frac{1}{16}$  to  $\frac{1}{3}$  mm. in diameter, and the older ones are deep black from the discharged spores, which often lie in a crust over the spot.

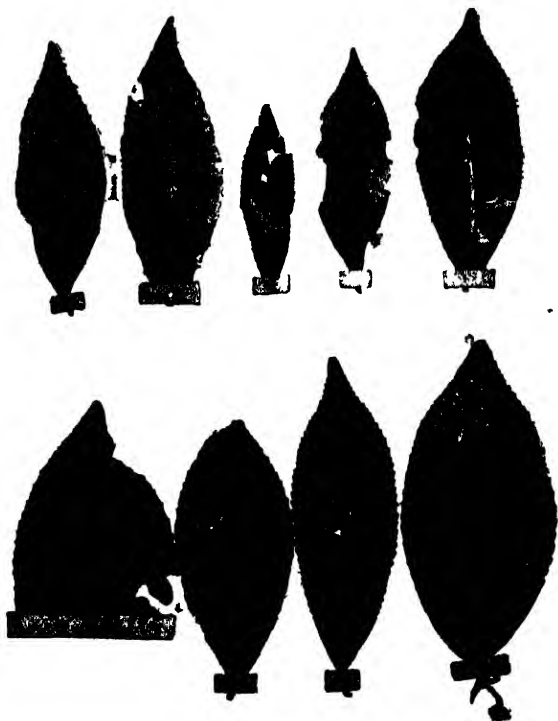


FIG. 184. Grey blight of tea (*Pestalozzia Theae*): affected leaves from different localities in India.

The old patches of diseased tissue dry up, become very thin, and break away easily.

The mycelium of the parasite extends between the cells of the leaf, throughout the discoloured part, the translucent margin marking the area of recent extension into the healthy tissues. The hyphae are exceedingly fine and difficult to see, sparingly septate, and colourless. Underneath the upper leaf epidermis, they collect into masses which develop into bowl-shaped, thin-walled spore-cases (pycnidia), the basal wall of which is

-distinct, while the lateral and top walls are slender and rather obscure. *Pestalozzia* is usually placed in the Melanconiales, but, as has been noted by several observers, if the presence of pycnidia similar to those found in the tea parasite should prove to be general in the genus, it will not be possible to retain it in this order of fungi.

On the inner wall of the lower half of the pycnidium, a layer of spore-bearing cells occurs. From each of these, a stalk grows out into the cavity of the pycnidium and terminates in a spore. When ripe, the spores are detached with the stalk, which remains as an appendage at the base of the spore. At the same time, the epidermis and the slender top wall of the pycnidium underlying it become raised up and then ruptured by pressure from below, opening outwards in a crack of irregular (often, tri-radiate) shape, through which the spores are liberated in such quantities that they collect in little black crusts round the mouth of the pycnidium.

The individual spores are rather striking objects. They are spindle-shaped, divided by four septa into a row of five cells, of which the three central are dark coloured while the other two form a kind of colourless cap at each end. From the lower end, the persistent stalk on which the spore was borne projects as a slender tail. At the opposite end, the end cell grows out into three (or rarely four) colourless, thread-like appendages (cilia) of considerable length, which are very possibly an aid in securing the dissemination of the spores by the wind.

The whole spore measures from 22 to 30 by 5.5 to 7 $\mu$ , the coloured part being 17.5 to 21 $\mu$  long and, therefore, less variable than the hyaline end cells. The persistent stalk is rigid, straight or curved to one side, and from 7 to 11 $\mu$  long by less than 1 $\mu$  broad. The cilia are usually as long as, or longer than the spore, and may be up to 40 $\mu$ , the maximum breadth at the base being slightly over 1 $\mu$ , and the free end terminating usually in a blunt knob.

The germination of the spores is peculiar. Usually only the lowest of the three coloured cells gives a germ-tube. It swells up, becomes nearly round, marked by a light ring round the middle, and then puts out a germ-tube, or rarely two, from the sides. Its further development has not been followed.

The blight is said to be endemic in many gardens in north-eastern India. Sometimes it appears to become epidemic and spread goes on until nearly every bush in large plots becomes injured. In other cases it seems only to attack old bushes—generally the weaker ones in a plot—and go no further; very much of the regular grey blight is, of this type. It usually commences on one side of a bush, very often on the same side of all the bushes in an affected plot, probably an indication that it is

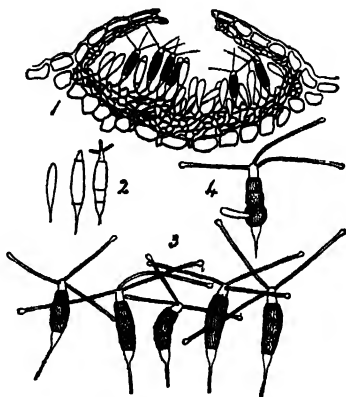


FIG. 185. *Pestalozzia Theae*: 1, section through pycnidium,  $\times 150$ ; 2, stages in development of the conidia,  $\times 430$ ; 3, mature conidia,  $\times 430$ ; 4, germination of a conidium,  $\times 430$ .

conveyed by the wind. It may then work round or over the bush, until every leaf is killed. One of the worst forms of attack is when the infection occurs near the base of the leaf, involving only the base and the stalk. In such cases, the leaf quickly withers and falls off. It is said that the bush is sometimes killed, but this seems to be doubtful. The destruction of leaf is, however, often sufficient to damage the bush seriously, all the leaves on one side, or perhaps all round the edge, being sometimes lost. In Ceylon, it has been reported to attack the stem at times, having been found on stems up to one inch thick and also, more frequently, on the young half-shoots left in plucking.

It is far from certain that all the injury attributed to grey blight is really the work of this fungus. In particular, a serious form, seen in Bishnath District and elsewhere, in which, when the diseased leaf touches a healthy one, the two become cemented together by mycelium extending from leaf to leaf (as happens also in thread blight), is very possibly a distinct disease. In many cases, too, it seems possible that insect injury is responsible for certain misnamed forms of grey blight.

Amongst the causes which predispose to attack are old age of the bushes; water-logging, or a sour, undrained soil, or the conditions found in deteriorated "bheels" in Sylhet; and curiously enough, in the opposite direction, exposure on dry, bare ridges. Both in India (Darjeeling) and Ceylon, grey blight has been observed to be very harmful to tea in exposed situations, where the soil is worn and poor and the bushes wind-swept. Attacks of "red spider" make the leaves very susceptible, hardening them before their time, whereas the young and succulent leaves are not attacked. Over-plucking is another cause. The essential factor in all these cases is the weakening of the vitality of the bush due to some previously existing cause, at the time when infection with the parasite occurs. This period is said in Ceylon to begin after the early April rains, but to be little marked until the onset of the monsoon, when infection rapidly increases, reaching its height about August. But it is said that a good flush of leaves appears when the monsoon comes to an end, even on bushes previously almost denuded of foliage, and this is, no doubt, one of the reasons why the disease is not much feared in Ceylon. There is little difference in the susceptibility of different varieties of tea.

The treatment is chiefly in the direction of reducing the supply of infective material early in the season. By itself, it is probable that spraying against grey blight would not usually repay cost; spraying need not be considered, therefore, except in so far as it incidentally checks

the disease when carried out against more serious affections. It has been suggested that in those, now comparatively rare cases, where a block cannot, for any reason, be pruned and cleared out during the winter, it may be sprayed twice with Bordeaux mixture, once in the cold weather and again in a spell of fine weather in April or May, to check blights generally.

In most cases, nothing more is needed than clean pruning, together with the collection of all diseased leaves. The prunings and leaves should then be burned or thoroughly buried. Removal of blighted leaves may, if necessary, be continued through the spring, and there are cases on record of successful action during the plucking season, the pluckers having been provided with separate bags for blighted leaves, which were paid for at the same rate as the leaf for manufacture. Early action is, however, most important; if nothing is done until the middle of the season and the bushes are then seriously infected, there is little hope of success.

Much of the grey blight seen is, undoubtedly, a symptom of weakness from some other cause, and the best treatment is often to search out this cause and remove it as far as possible.

In a peculiar form of leaf disease found in certain parts of Assam, both *Colletotrichum Camelliae* and *Pestalozzia Theae* seem to combine to cause the injury, which is known as "rim blight." The leaves wither and shrivel round the edges, the decayed portion being chocolate-brown in colour and quite brittle, so that it crumbles away, leaving the margins corroded in a characteristic fashion. This progresses until the whole leaf withers. It spreads faster than the cases of infection by either fungus alone, but is said to be rather a special blight of certain varieties of tea. It has also been recorded in Ceylon.

It is said that the tea *Pestalozzia* is identical with one which causes a common leaf spot on Heves. In view of the inaccurate determinations of the identity of the former, it is evident that this requires confirmation.

A good many years ago a leaf disease of tea in Cachar was attributed to the attack of a fungus named *Hendersonia theicola* Cke. More recent examination of the specimens preserved at Kew have, it is stated, shown that this is only an immature or poorly-developed form of *Pestalozzia*.

**Leaf spot** (*Cercospora Theae* van Breda).—It is very common, towards the end of the season, to find the leaves of tea, in the north-eastern districts, bearing the small circular spots caused by this fungus, but without any appreciable damage being caused to the bush. It was

first described in Java and is also known in Ceylon, in both cases being considered of as little importance as in India.

The older leaves only are attacked. Little reddish-brown spots appear, which rapidly penetrate the leaf and show on both surfaces. They spread but little laterally, usually not exceeding 2 or 3 mm. in diameter, this limited growth being in strong contrast to the other leaf fungi previously described. After a time, the centre of the spot dries up, becomes very thin, and turns white. It then often falls out, leaving a clean-cut, circular hole, similar



FIG. 186. Shot hole of tea (*Cercospora Theae*): leaf,  $\times 1\frac{1}{2}$ ; conidiophores and conidia,  $\times 430$ .

to the well-known shot hole disease of many temperate fruit trees.

The mycelium of the fungus is entirely immersed in the leaf, only the fructifications appearing on the surface. These consist of densely crowded bundles of conidiophores which emerge through the stomata of the under surface of the leaf. The conidiophores are short, with the angular heads in the sides commonly found in *Cercospora*, *Helminthosporium*, and allied genera, and smoky-brown in colour. They bear long, slender, blunt-ended conidia, up to  $120$  by  $3\mu$  with 5 to 10 or more cross-septa, and of a very light brown colour. These fall off readily.

No case has yet been recorded where treatment was required.

**Thread blight (sterile mycelium).**—Although the remarkable disease known by this name has been under observation for many years, the exact nature of the parasite which causes it is still obscure. It is referred to in the Proceedings of the Agri-Horticultural Society of India in July, 1868, and is hence one of the oldest known tea diseases. Still, it seems to be confined to India, as it is exceedingly doubtful if the somewhat similar diseases which have been described in Java and Ceylon are really thread blight.

It occurs in all the north-eastern tea districts, and is said to be by no means confined to tea, but to exist on a large number of jungle and

cultivated plants in Assam, amongst those mentioned being bamboos, *Dillenia indica*, *Eriobotrya japonica* (the loquat), *Achras Sapota* (the sapodella), and mango, as well as the common hedge plant, *Duranta*. It is necessary to bear in mind, however, that there are probably several distinct fungi likely to be mistaken for thread blight, and that, as they are ordinarily sterile, it is only by inoculation experiments that the range of hosts of any one species can be determined. It has been reported, for instance, that infected leaves of *Dillenia indica* can communicate the blight to tea, and experiments with other plants might usefully be made so as to get an accurate list of those on which the fungus can live. Ferns and herbaceous weeds growing in contact with affected bushes become attacked by direct extension of the parasitic mycelium from the tea.

The attack becomes visible in the early part of the season by the appearance of white threads or strands of fungus tissue, which pass along



FIG. 187. Thread blight of tea : affected shoot showing mycelium on stem and under surface of leaf on left,  $\times \frac{1}{2}$ .

the branches and spread out into fine, web-like films on the under surface of the leaves. These threads appear to arise frequently from the older wood, and ascend the younger branches to reach the leaves. Later on, it is common to find the mycelium starting from the outer part of a bush and passing down the twigs to the first fork, when it usually turns up again. Lateral spread occurs freely wherever the leaves or twigs are in direct contact, the mycelium sometimes binding the parts closely together ;

in great humidity it may even cross spaces up to quite half an inch. Rapid spread is always in the form of the white mycelial strands, the leaf films being of slower growth and seeming to serve chiefly for nutrition. Under ordinary circumstances, the white strands, though soft, are firm and smooth on the surface, but in very moist air they become fluffy and of a consistency resembling cotton wool, while the diameter increases considerably. At the base of a petiole or branch, they often enlarge to form a sort of cushion. On the leaves, too, there are sometimes condensations into thicker masses or strands, here and there, in the films. Below the smooth-barked portion of the younger stems, the fungus becomes difficult to trace. Distinct strands can be often found as far down as the junction with the old wood towards the centre of the bush, but are rare on those main stems not removed in heavy pruning. A common condition, where the disease is of long standing and attempts have been made to cut it out, is to find an old, pruned-back stump, 6 to 12 inches in length, itself free from fungus, though often stripped of bark from a previous attack, and, arising from it, a number of infected young shoots. On the leaves, the film ordinarily arises from the base and spreads out fanwise at first. When the margins are reached, growth is sometimes continued around them to the upper surface, but direct attack on the upper surface has not been observed, and the growth here is always limited and does no apparent harm. On all green parts, the threads and films of the fungus can be lifted off without difficulty when wet.

The effect on the leaves is that browning and death of the leaf cells sets in, some time after the film spreads out on the under surface. This effect is, for a time, sharply limited to the part covered by the fungus, but, especially when the attack begins from the base, the rest of the leaf soon withers, and it falls off. It is often held for a time by the threads which join it to the twig, and one of the most striking appearances seen in this disease is the mass of hanging leaves, suspended, rotting, by white threads in the middle or at one side of the bush.

The rapidity with which leaves become brown and rot, when the fungus appears on their under surface, is striking. As there is no penetration of the tissues, it has been suggested that this effect is produced by stoppage of the orifices of the stomata by the film. That this is not the explanation is, however, proved by the local rotting produced when even a small patch of the leaf surface becomes covered, and also by cases where a green herbaceous stem of some weed is attacked



and turned brown at one point in its length, where it touches a diseased leaf or twig, the parts above and below remaining green for a while. In these cases, the mechanical coating of the surface can do no harm, as the air spaces communicate freely with unaffected parts. There seems to be no reason to doubt that the fungus obtains its nourishment from the browned parts, even though it does not penetrate them, such cases are not common, but there are several which are hard to explain in any other way. The actual feeding process may be by osmosis through the walls, but nothing is known of its details.

The mycelium consists for the most part of long slender sparingly branched hyphae, of uniform size 2 to 3  $\mu$  in diameter. Where they concentrate in little cushions or strands on the leaf the structure is as follows. The outer surface is composed of long, septate hyphae lying mostly parallel to one another and with numerous clamp connections and anastomoses. Some filaments turn up into the air when kept very damp causing the surface to look fluffy. Further in, the density increases the hyphae lying more closely together and being somewhat thinner. In direct contact with the leaf the structure changes. The innermost hyphae give out numerous short branches which intermingle to such an extent as to give almost a cellular (pseudoparenchymatous) structure. In the older cushions and strands, the surface consists of a dense layer of branched hyphae with numerous anchor cells like those of the cobweb disease of coffee (p. 486). In the inner layers the branches arise chiefly from one side only of the hyphae, and are very varied in size and shape, from mere lateral pouches in the wall, to forked filaments which may unite by anastomoses with neighbouring branches. The filmy part of the mycelium consists of a very fine uniform, web like layer of hyphae. No trace of penetration of the epidermis has been detected, and the fungus can be lifted off when wet without breaking it. It seems probable that the peculiarly branched, inner layer described above forms a sort of hold fast or appressorial layer, though the cuticle does not seem to be indented or marked by its branches. On the petiole and green part of the stem, the strands are composed of hyphae similar to those of the outer layer on the leaf described above, and entirely superficial. Lower down, however, the fungus enters the tissues of the stem. This commences sometimes immediately after cork formation begins. The outer cells of the cortex, external to the cork cells, dry up and are readily penetrated by the hyphae. Branches pass down through the cork layer, which as already mentioned under red rust (p. 417) is not continuous when first formed, and accumulate in the cortex below the young cork. The result is the gradual destruction of all the tissues external to the hyphae. The outer bark splits in longitudinal strips, which at first gape, then roll up and separate off. New cork is formed deeper in, and the process is repeated, until the bark is often entirely destroyed and the wood exposed. The hyphae have been traced as far in as the cambium. The extent to which the injury to the stem is responsible for the damage caused by thread blight is not known, it is probably not so great as in red rust, but the latter is a notable warning of the very serious injury which can be produced by a similar form of attack.



FIG 188. Thread blight of tea structure of a mycelial strand from a leaf.

No fructification is known in this fungus. The examination of dried specimens sent to England in 1898, led to the belief that a little, reddish, club-shaped fungus found on twigs attacked by thread blight, was the reproductive stage of the latter. It was named *Stilbum nanum* Massée. Further investigation of living material in Assam has shown that the mycelium of the *Stilbum* is distinct from that of thread blight, and that each fungus commonly occurs without the other. It is now generally accepted that the thread blight fungus is probably a Basidiomycete, as suggested by the clamp-connections and its resemblance to a group of forms of considerable interest as plant parasites, the most notable of which is *Corticium Koleroga* on coffee. In Java, a very similar tea parasite has been named *Corticium Theae* Bern., but is described as being pale rose-coloured on the leaves and sometimes also on the stems and as not penetrating the cortex of the latter, and is, therefore, probably distinct. In Ceylon, a disease caused by a species of *Crepidotus*, and characterised by the formation of white strands on the leaves and twigs, has been described on the nutmeg. This fungus belongs, however, to quite another section of the Basidiomycetes than *Corticium*, and from the description and illustrations published it appears to be different from thread blight. Still it has also been found on tea twigs on a few occasions, and is apparently believed in Ceylon to be identical with the Indian disease.

Infection occurs, so far as is known, only by leaves or twigs carrying the fungus being blown from diseased tea or from the jungle. The latter is said to be very common. At one time, it was suggested that soil infection might also occur and the fungus grow up under the bark to the younger wood, but attempts to produce the disease in this manner failed. In such cases as appear to be examples of infection from the ground, it is probable that some of the previous year's mycelium has remained in the older stems not removed in pruning, and has started afresh on the new shoots that arise from these stems. In newly-infected bushes the start from a foreign leaf somewhere in the periphery of the bush can sometimes be actually seen. This accounts for the sporadic nature of the cases, which are generally scattered on odd bushes here and there and not in definite patches. Such patches are only found sometimes in the neighbourhood of jungle where there is much blight, and every bush is liable to infection from without.

Unlike red rust and several of the other disease of tea, this blight is not more prevalent on unhealthy or weakly bushes. The only predisposing causes reported are excessive shade and high atmospheric humidity.

The treatment usually adopted is to cut out all the parts affected, at the pruning season. Since this has been generally done, the blight, which sometimes used to be found on from 20 to 50 per cent. of the bushes in a block, has ceased to do much damage. It is important to burn the affected parts, as the mycelium goes on growing for quite a long time after removal from the bush, if kept moist. It is even said that buried prunings have conveyed the disease to the roots, but this is certainly rare.

The cutting out is ordinarily done at pruning time, which is infinitely the most convenient time for doing it, but not necessarily the most effective. Probably less would be missed if the bushes could be gone over fairly early in the rains, when the white threads are found running up single shoots from the older wood. These cases are not dealt with until December or January, by which time much of the bush may be affected and severe cutting necessary.

In some gardens, the affected bushes are left alone in the general pruning and stand up conspicuously; they are then gone over by experienced men and all diseased wood removed.

The excellent results obtained by painting with fungicides such as lime and sulphur, after a moderately hard pruning which removes the diseased leaves and smaller twigs and opens up the heart of the bush, have led to this method being widely used. The lime and sulphur mixture is prepared by slaking  $7\frac{1}{2}$  lb. fresh quicklime with water, then mixing with this  $2\frac{1}{2}$  lb. sulphur, then adding enough water to make up to 10 gallons, and finally boiling in an iron vessel until the liquid becomes orange in colour. It is applied to all parts of the stem that show signs of disease, with a brush or swab of any convenient material. The exact way in which this acts is worth examining, as it would appear that the part of the fungus on the older twigs under the bark ought to be out of reach of any direct-acting substance. Possibly the wash still adhering to the bark when growth is renewed and the strands appear at the surface is sufficient to kill them. In certain cases, bushes have been found in which practically all the young shoots from a cut-back stump were infected by growth which clearly started from within the bark of the old stems, though these still bore traces of the lime and sulphur mixture applied some seven months earlier.

In the majority of cases where the disease is taken early, cutting out alone is often sufficient. Where, however, it has got a good grip of the bush and cutting out completely means much mutilation, the lime and sulphur treatment is said to have great advantages.

**Sooty mould** (*Limacinula Theae* Syd. & Butl.).—In tea bushes infested with scale insects, a black fungus, related to that already described under sugarcane (p. 411), is often found. Like the latter, it grows in the sugary secretion ("honey dew") of certain insects and only accompanies these insects, disappearing when the plants become free from them. The injury caused by the sooty moulds is slight, and entirely

restricted to interference with the proper lighting of the cells. Their dense, black structure reduces the amount of light reaching the chlorophyll, and consequently diminishes the activity of the latter in manufacturing food.

The species on tea in Darjeeling is a distinct one, not hitherto known elsewhere. Whether the same species occurs throughout the tea districts is not certain, as those forms previously examined have been named

*Capnodium Footii* B. & Desm., a name which probably covers a number of different species.



FIG. 189. Sooty mould (*Limaecula Theae*) and black blight (*Asterina Camelliae*) of tea: 1, small leaf and twig with sooty mould,  $\times \frac{1}{2}$ ; 2, pyrenidia and pyrenospores of same; 3, a sterile hair from the mycelium of same; 4, a perithecium of same with asci; 5, an ascospore; 6, leaf with black blight,  $\times \frac{1}{2}$ ; 7, a perithecium of same underneath a network of broad hyphae; 8, ascus, and 9, ascospore of same.

broader, about 80 by  $30\mu$  in diameter (Fig. 189, 2). Both forms contain numbers of very small, colourless, short, cylindrical spores,  $2\frac{1}{2}$  to 3 by  $1\frac{1}{2}\mu$  in diameter. Perithecia are less common. They are spherical or somewhat flattened, 160 to  $225\mu$  in diameter, with a mouth in the middle of the free side, and contain ovoid, 8-spored asci with very short stalks (Fig. 189, 4). The spores are oblong, rounded at the ends, hyaline, and divided by long as well as cross septa, generally 5 of the latter, and one of the former (Fig. 189, 5). They measure 25 to 33 by 9 to  $1\frac{1}{2}\mu$ .

The Darjeeling sooty mould forms a black crust on the upper surfaces of the leaves and on the twigs (Fig. 189, 1). This crust is membranous for the most part, but in places thickens into rather woolly masses, largely composed of various kinds of fructifications.

The mycelium is purely superficial, and consists of dark brown, septate hyphae from which arise, here and there, clusters of black, spine-like hairs, pointed at the top, and measuring up to 130 by  $8\mu$  (Fig. 189, 3). Conidia are borne on lateral branches of the mycelium, and belong to the type of the imperfect genus *Triposporium*, each spore being composed of 4 radiating arms. In the woolly parts, pyrenidia are found in great numbers. They are of two kinds; some long (up to  $300\mu$ ), slender, and slightly swollen in the middle (up to about  $22\mu$  across); others short and

The treatment is to free the bushes from the insects (generally scales) with which the fungus is associated and for the presence of which it often serves as a useful warning.

**Black blight** (*Asterina Camelliae* Syd. & Butl.).—This black leaf-fungus differs from the last in that it is not associated with insects and is probably parasitic, though mostly superficial. It is also of more limited growth, not extending over the whole leaf as sooty mould frequently does. It has been found in Assam only, and seems to be uncommon and practically harmless.

The fungus forms very small, radiating crusts (Fig. 189, 6), united in patches of irregular shape by a loose network of large, dark, reddish-brown hyphae, which lie on the crusts and extend across from one to another (Fig. 189, 7). These hyphae are 9 to 12 $\mu$  broad and their branches arise often in pairs on opposite sides of the hypha but sometimes are alternately on one side and the other. Other alternately situated, knob-like protrusions, known as hyphopodia, also occur, being nearly round or sometimes tri-lobed, and 12 to 16 $\mu$  broad. The radiate crusts are perithecia, up to 340 $\mu$  in diameter and opening when ripe by radiating cracks (Fig. 189, 7). They contain a small number of ellipsoidal asci, measuring 70 to 100 by 25 to 35 $\mu$  and containing, apparently, each 8 spores (Fig. 189, 8). The spores are long elliptical, brown, unseptate, deeply constricted at the septum, with slightly unequal cells and rounded ends (the lower sometimes obtusely angular), and measure 30 to 40 by 16 to 18 $\mu$  (Fig. 189, 9).

**Canker.**—This is perhaps the most important tea disease left for investigation. Nothing is known of its cause, not even whether it is due to a fungus. In some cases, the stem cankers caused by red rust have been confused with it; in others, it seems to be associated with certain insects (scales) and perhaps a mite. It is found in Assam, the Duars, and the Wynaad, and has been known for a good many years but is probably increasing. In Cachar, where it is a serious disease, it is said to be worst on deteriorated "bheels"; elsewhere it is found chiefly in badly drained land. It does not seem to be known outside India. It occurs on the stem and twigs, causing swellings and cracks in the bark and, ultimately, open wounds down to the wood (Fig. 24). These wounds may ring the larger stems and kill all the parts above.

No parasite has as yet been found constantly associated with the disease, and though it was formerly stated to be allied to the well-known canker of fruit trees caused by *Nectria ditissima* in Europe, this appears to refer to only one form of it, prevalent especially in Darjeeling. The *Nectria* in these cases is more closely allied to *N. cinnabarina*.

The only treatment known is to cut out all the diseased wood at pruning time.

**Seedling disease.**—A disease of young seedlings has been signalled on several occasion in India, Ceylon, and Java. The appearances are so characteristics and similar in the three cases that the cause is probably identical in all.

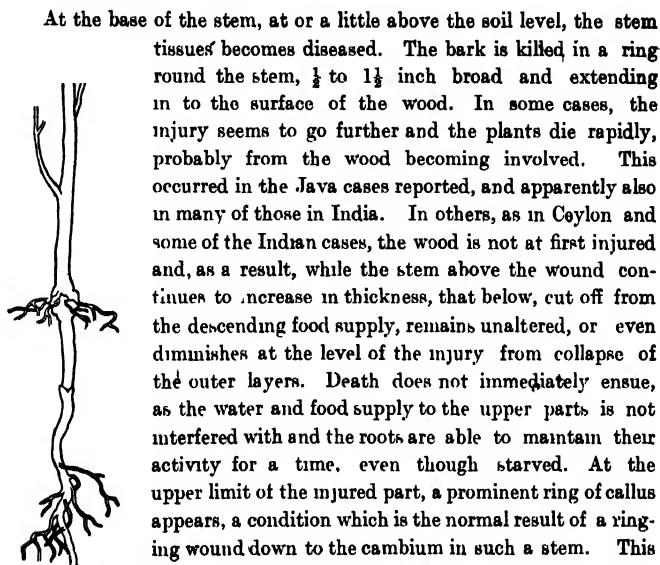


FIG. 190. Tea seedling disease (diagrammatic). (After Hope and Carpenter).

In those cases where the attack rapidly involves the wood, new branches may spring from below the seat of injury, and either replace the killed primary shoot or are themselves killed off in turn.

Both in India and Java, the original cause of this disease has been assigned to alterations of high humidity and great heat. The conditions were most closely examined in India, and it was found that the disease occurred in a season in which there was, first, a long drought with considerable heat towards the end, then, continuous heavy rain for about a fortnight, and then, several extremely hot days. During these, the disease became evident. No parasite was detected, the roots were quite normal, and the whole trouble was attributed to the climatic changes. In Java

and Ceylon, on the other hand, a fungus has been found in the diseased parts and is considered to be the direct cause, Java opinion holding, in addition, that abrupt climatic changes prepare the way for the attack. Further study is, therefore, necessary, before the factors concerned in this disease can be assigned their true worth.

As precautions advisable in Upper Assam to check the loss of seedlings from this cause, it has been recommended to plant the nursery early, so as to allow the seedlings time to establish themselves ; to supply shade, and water, in view of an early and prolonged drought ; and to remove adhering earth round the base of the stem, when weeding the plantation.

**Tea seed mould.** -It is a common experience to find the inside of the mature tea seed affected with a mouldy growth, while the surface appears sound. The mould is found usually on the rudimentary cotyledons, sometimes without causing much apparent damage, sometimes reducing the cotyledons, and even the embryo, to a shrivelled mass. In such cases, the value of the seed is greatly lessened, owing to the high percentage of failures to germinate.

The moulds found are common species, *Penicillium* and the-like, several of which are similar to those so often found causing the decay of ripe fruit. These may be looked on as very weak parasites, able only to attack cells gorged with sugary juices or otherwise of low vitality.

It was long a puzzle how such moulds gained access to the inside of the seed, and how they were able to grow there, supposing that the seed was healthy to start with. A very reasonable explanation has now been found. A large sucking insect, *Pæcilcoris latus*, has been observed to live on the juices of the tea fruit, which it obtains by deeply puncturing the rind by a special apparatus in the beak. Both immature and ripe fruit are thus attacked and the juice sucked out, often without, in the former case, preventing the seed from continuing its development. When the beak is withdrawn, no doubt a little moisture from exuded cell sap remains at the puncture. Air-borne spores falling in this would readily germinate, and their germ-tubes very likely grow down the wound to the inside. Here they would encounter cells weakened by the loss of sap, or even sometimes killed, and would be able to thrive according to the extent of the injury. To test this, a large number of these tea seed bugs were fed on unripe fruits and the seed subsequently examined. All those opened were

found to contain fungus, in most cases at the expense of the proper contents, which were reduced to shrivelled remains, but sometimes only in a small, well-defined patch on one of the cotyledons. Seeds of the latter type would doubtless have reached maturity and have appeared perfectly sound from the outside.

Since this view was suggested, the insect is regarded as a serious pest in seed gardens, and measures to collect and destroy it have been recommended.

Coffee berries are said to suffer in a similar manner, and it is suggested that an allied insect, *Bagrada picta*, may be responsible.

---



## CHAPTER XIII.

### COFFEE.

(*Coffea* spp.).

THE diseases of coffee in India are even less completely known than those of tea, if we except one or two of the commonest and most destructive. Thus no root disease has been fully investigated, though at least two serious maladies of this type occur in southern India. No stem diseases have been described, though one has caused a considerable amount of damage in the Nilgiris, and others are known to attack coffee commonly in neighbouring countries and probably occur in India. Even with regard to the leaf diseases, which are better known than any other class on this crop, we have to turn for much of our information to work done in other countries, and the local conditions which influence these diseases, and even their distribution in the different coffee districts, are not as a rule recorded and seem to have been little investigated.

Yet coffee is the classical example of the ruin of a planting industry through an epidemic disease of cryptogamic origin. After the middle of the last century, it was, for a time, the mainstay of the prosperity of Ceylon. The production about 1870, towards its maximum, was nearly 850,000 cwt., worth on the spot about four millions sterling. A year or two prior to this, a fungus had been observed on the leaves, which was new to the planters and, as it proved, new to science also, and somewhat of a scientific curiosity. It spread with great rapidity, and by 1872 was recognised to be doing considerable damage. How great the damage was, may be judged by the fact that in the ten years following its appearance, it has been calculated that it caused a loss of from 12 to 15 million sterling, and reduced the average yield per acre from  $4\frac{1}{2}$  cwt. in 1870 to less than 2 cwt. in 1878. Owing to the opening of many new estates, the total export remained considerable until after 1880, but then rapidly fell off, in 1884 being 176,000 cwt. only. Coffee planting had ceased to be profitable; the area under the crop shrank rapidly; and though, on its ruins, the present flourishing tea industry of the colony arose, the

economic dislocation was, for a time, the cause of much hardship to the planters and much uneasiness to the Administration.

**Rust or leaf disease** (*Hemileia vastatrix* B. & Br.)—The coffee leaf disease was first noticed in Ceylon in 1868. The following year it was recorded in Mysore. It was found in Sumatra and Java in 1876, and reached the Fiji Islands about three years later, coming, it is said, direct from Ceylon with a box of coffee seeds. It was probably in Natal about 1878, Mauritius about 1880, Réunion in 1882, and the Philippine Islands in 1885. It has been in Madagascar since at least 1886, but is stated to have been really introduced in 1872 or '3 by a Ceylon planter who came to examine coffee growing in that Island. It was found in Tonkin in 1888, and reported in Malaya and Borneo in the same year. In 1894, it was in Samoa and reported from German East Africa, but has since been found on old leaves of *Coffea arabica* collected in the latter colony in 1885 and there is no reason to believe that it may not have been in east and central Africa as long as the species of *Coffea* found wild and semi-wild in that part of the world. It now occurs in most colonised parts of east and central Africa, including the Congo, but has not yet been signalled from the West Coast. The New World as a whole is free from it, and also, apparently, Australia and Hawaii, but the New Hebrides and New Caledonia have only recently been reached—the latter in 1910 or '11—so that extension is still going on.

The origin of the disease in Ceylon has given rise to much discussion. Some have believed that it came from *Coffea travancorensis* or *C. liberica*: but, as regards the latter, the fungus has not yet been found on the West Coast of Africa, whence this variety originated; while if endemic in South India and Ceylon, the home of the former, one would expect the disease to be known in these areas at an earlier period in the history of their coffee cultivation. Others suggest that it may have spread to coffee from some jungle plant. Several other species of *Hemileia* have, in fact, been since discovered on various plants, and some of them are scarcely distinguishable from *H. vastatrix*. This possibility cannot, at present, be entirely excluded, but attempts in South Africa to infect coffee with species such as *H. Woodii* from *Vangueria infausta*, and *vice versa*, have entirely failed. Further, the only closely allied species so far found in India is *H. Canthii*, which is not uncommon on leaves of *Canthium* in Mysore and also occurs in Ceylon. If the coffee parasite came from this plant, it is difficult to explain its tardy appearance, a good many years after coffee growing had been undertaken. It is, perhaps, most probable

that *H. vastatrix* is endemic in some of the countries of origin of the cultivated varieties, as has been suggested to be the case in east and central Africa, where it has been discovered on the native plants of *C. robusta* found in the Victoria Nyanza region and elsewhere, and also on a wild or semi-wild species of coffee in German East Africa. Though, in the latter case, the fungus was first described as *H. Woodii*, re-examination has shown that it cannot be distinguished from *H. vastatrix*, and there is no evidence that any other *Hemileia* occurs on coffee. It seems, therefore, reasonable to suppose that the rust, originating in Africa, has spread, as so many diseases have, through human agency, by the transport of living plants or other contaminated material from one district to another, and ultimately crossed the ocean to Ceylon.

The disease is confined to the leaves as a rule, only rarely being found on the fruits and, perhaps, near the tips of very young branches. The first symptom is the appearance of small, yellowish spots, a millimeter

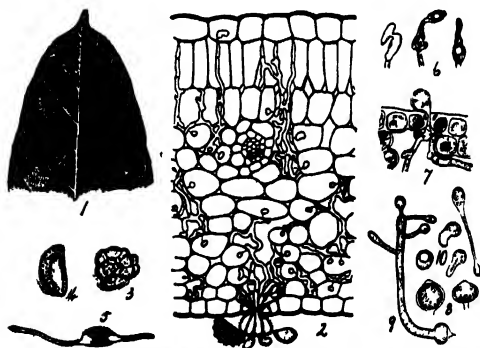


FIG. 191. Coffee leaf disease (*Hemileia vastatrix*): 1, under surface of affected leaf,  $\times \frac{1}{4}$ ; 2, section through same showing mycelium, haustoria, and a spore-cluster; 3, a spore cluster seen from below; 4, a uredospore; 5, germinating uredospore; 6, appressorial swellings at tips of germ-tubes; 7, infection through stoma of leaf; 8, teliospores; 9, teliospore germinating with promycelium and sporidia; 10, sporidia and their germination. (2 after Zimmermann, 3 after Delacroix, 4-10 after Ward).

or two in diameter, on the under surface of the leaves. As the spot enlarges, the colour becomes more intense, and when it is about 3 mm. in diameter, the surface becomes covered with an orange powder composed of the spores of the parasite. The spot may enlarge subsequently up to  $1\frac{1}{2}$  cm. (Fig. 191, 1). On the upper surface there is no discoloration

at first, but, later on, livid or brownish patches appear in corresponding situations to the spots below. At a late stage, the centre of the spot on the under surface becomes grey or brown, the margin alone remaining yellow. The orange spore-powder is not found on the upper surface. The spots remain rounded for the most part, but the temporary check caused to their growth by the veins of the leaf, and also the union of adjacent spots, often lead to irregularity in shape. When the attack is severe, the whole leaf may turn brown and dry up.

The attack usually sets in soon after the advent of the south-west monsoon, and the worst damage is usually caused, in South India, during the rains, though an attack in the dry season (December–January), which has a deleterious effect on the ensuing blossom, sometimes occurs. The young leaves may be attacked as soon as they appear, but as there is a period of some 14 days' incubation before the spores develop, the youngest leaves show no sign of the fungus at first. Mature leaves are less readily infected than the young. Plants of all ages are attacked, even seedlings in the cotyledon stage.

In bad attacks, the diseased leaves are shed in quantity. In the Philippines, it has been recorded that the branches of infected trees only kept 2 or 3 pairs of leaves instead of the usual 6 or 8. In Ceylon, it has been similarly observed that sound leaves remain on the trees for 18 to 20 weeks or more (Java observers say for as many months), while leaves with numerous spots of disease fall in from 6 to 11 weeks.

The injury caused to the bush at the first attack is usually not very considerable. The assimilatory function of the affected leaves is interfered with, with a consequent reduction in the nutrition of the bush. Further, an attempt is made to put out new leaves to replace those lost from the disease, and this throws an extra strain on the bush. The result is a diminution in the crop for the time being; but the parasite disappears so completely at certain periods, and in certain districts the bushes, when relieved from its influence, so readily put forth new growth and bear considerable crops, that in the earlier days confident hopes were entertained that the mischief would soon entirely pass away. These hopes were doomed to disappointment. It was found that sooner or later, if the locality were favourable to the activity of the parasite, the attacks recurred; many of the bushes, particularly the older and less vigorous, became unable to mature their crop, the berries remaining small and often failing to ripen; the stronger bushes were gradually weakened by recurring attacks; and the total crop progressively fell away.

In some cases, even the first attack had disastrous results; in the Philippines, a reduction to 35 per cent. of the estimated crop occurred a few years after the first outbreak, and by 1890 the industry had practically ceased to be profitable, while in New Caledonia, some plantations reported a loss of half the crop in 1912, a year or two after the disease appeared. The more typical experience, however, appears to be that of Java, where the attack was slow and progressive and seems to have reached its maximum in the early years of the present century. The average annual yield in the Government plantations fell from about 60,000 tons between 1879 and 1883, to about 40,000 tons between 1884 and 1888, and the fall continued, the total crop in 1908 being less than one-third of the yield formerly obtained. It is, indeed, fairly certain that the industry was only saved from utter destruction by the introduction of the newer resistant varieties of coffee to be mentioned below. It is difficult to obtain any estimate of the losses caused in India; in certain localities coffee cultivation has been almost abandoned, but, on the whole, it seems doubtful if the epidemic equalled in severity that in other countries; leaf disease rarely kills the bushes and the loss of crop varies greatly according to the locality, the nature of the season, and the age and vigour of the plants.

The rust is usually found in the uredo stage only. Teliospores have been discovered in Ceylon, but they appear to be rarely produced and have not been encountered in other countries, except in the form on a wild species of coffee in German East Africa. The æcidial stage, if there be one, is unknown, for though an *Æcidium* occurs on coffee in Mysore, it has probably no connection with *Hemileia*. There is abundant evidence that this stage in the life-history of the fungus is no more essential than in many other rusts, notably *Puccinia triticina* and the other wheat rusts in India.

The mycelium is found in the discoloured spots only, ramifying in the form of moderately thick, usually colourless hyphæ between the cells of the leaf parenchyma (Fig. 191, 2). Septa are found at long intervals, and haustoria penetrate the cells as little swollen sacs communicating by a narrow tube with the mycelium outside. The cell contents are altered, the chlorophyll fades, and the protoplasm and its inclusions become collected into a brownish mass or sometimes even disappear, to be replaced at first by sap and then by air.

The hyphæ, after a time, collect into masses in the air spaces below the stomata on the under surface of the leaf, and bundles of closely-adherent stalks emerge from these masses, through the stomatal openings, to the surface of the leaf. Each stalk swells up, soon after it emerges, into a hyaline enlargement, which is cut off by a septum at the narrowed base (which forms a kind of pedicel or sterigma) and becomes a uredospore. New spores bud off from the stalk lower down. Each stalk thus produces several spores attached by short sterigmata to its tip, which may be somewhat swollen to carry them. As age advances, the stalks become less distinct one from another, and form a compound column, at the end and sides of which a densely-crowded mass of sterigmata carry the spores in a head (Fig. 191, 2-3). The spores themselves are at first roundish, hyaline,

and smooth-walled; then become elongated and in shape something like a shallow segment of an orange, triangular but with broadly rounded corners in cross-section (Fig. 191, 4). The outer side is convex, the inner surface often concave lengthways and convex from side to side but sometimes almost plane. This is, in fact, due to mutual pressure of the spores, and hence varies according to their position in the cluster. As they mature, the contents turn orange-yellow, while the wall remains hyaline and is provided with prominent warts on the convex outer face, those at the edges being longer than the rest. The spores measure when ripe 30 to 40 by 27 to 30 $\mu$ , and soon become detached from their stalks.

The uredospores germinate readily in water, after an immersion of some hours (Fig. 191, 5). Two or three germ-tubes are usually formed, the wall being furnished with 3 to 5 germ-pores. The germ-tubes early turn orange by the passage into them of the contents of the spore, and are often provided with short branches. Appressoria are formed at the ends of the germ-tubes, sometimes cut off by a septum, sometimes not (Fig. 191, 6); and infection takes place by an infection hypha from one of these appressoria penetrating a stoma to reach the interior of the leaf (Fig. 191, 7). Owing to the scarcity of stomata on the upper surface, infection usually occurs underneath, where they are numerous; to infect the upper surface it is necessary to remove the epidermis first. This is one of the reasons why young leaves are the most vulnerable; while they are still inrolled, their under surfaces are more liable to be wetted and are also more easily reached by the spores. The extension of the mycelium is also more rapid within young leaves, owing to their tissues being softer and less resistant than those of older leaves. It has been found that if the leaf is kept too wet, appressoria are not formed and infection fails, the germ-tube remaining narrow and giving a long, branching hypha, which crosses the stomata without entering; but if the water dries slowly, the germ-tube is short and thick, appressoria early develop, and infection occurs. Long exposure to strong light hinders germination, but a short exposure of 20 or 30 minutes is advantageous: this effect is confined to the blue end of the spectrum, the red having no influence. Spores kept in the dark, germinate better than those exposed to strong light for more than an hour, but worse than those exposed for 30 minutes. If the spores have been allowed to dry, germination may be delayed, but it has been found that the dried spores preserve their vitality for several weeks, and they have been germinated in Europe after having been sent from the tropics. Extremes of temperature are unfavourable to germination, the optimum temperature being about 24°C.

After infection, a variable period passes before the spores are formed. The average of this "incubation" period is about 14 days. Very young leaves may bear spores after 9 days, if the surrounding conditions are favourable; while on adult leaves, the period may be delayed to 17 days. The orange patches are frequently most pronounced on the third pair of leaves, since the incubation period is usually long enough to allow two new pairs of leaves to unfold between the time of infection of the apical leaves and the appearance of the spores. The same spot may, however, continue to bear spores for many weeks.

The teleutospores of *Hemileia vastatrix* have only been found in living specimens by one observer in Ceylon, but they have also been observed on Ceylon and African specimens in the Kew Herbarium. They are formed in the same manner as the uredospores, amongst which they may be intermingled and the young stage of which at first they resemble. When adult, however, they are turnip-shaped, sometimes broader than long, and with an apical papilla (Fig. 191, 8). They are attached by a short stalk to the compound spore-bearing head described above as carrying the uredospores, but are only found at an advanced stage, when the leaf spot has ceased to spread and the spore-bearing heads have reached their full complexity. They then appear mingled with the uredospores, or if these have fallen, almost alone. They have the same orange-coloured contents as the latter, but the wall is smooth. Their size is about 30 by 25 $\mu$ . Germination occurs

by the growth of a promycelium from the apical papilla (Fig. 191, 9). This promycelium becomes 3-septate and bears 4 roundish sporidia on slender sterigmata, one from each cell. These sporidia germinate readily (Fig. 191, 10), but attempts to infect coffee leaves with them have failed and their further history is unknown.

So far, no variety of coffee has been found to be quite immune to the rust (though this is said to have been the case for at least several years with *C. congensis* var. *Chalotii* in Madagascar), but the degree of susceptibility differs greatly, not only from one variety to another but also within the individuals of the same variety. This fact is the basis of most recent work in the fight against Hemileia.

Besides the inherent variation in power to resist the disease, it was early found that its intensity in a given plantation depended from year to year on several factors, some controllable, some not. The final rapid destruction of much of the liberica variety in Java was hastened by three successive wet years, 1909 to 1911. Excessive rainfall is not in itself a cause of severe attacks, as is evident in Mysore where the Ghat districts suffer less from Hemileia than those of the Malnad, though the rainfall in the latter is less than half that of the former. It is probable that, as in many similar cases, the most favourable conditions for the development of the parasite are periods of overcast weather with high humidity and moderate rain, especially if broken by intervals of fine weather. The influence of the climate is strikingly illustrated in Réunion, where the severity of the disease has made it practically impossible to cultivate the arabica variety on the side of the island with a damp, steamy climate, while on the other side, where there is a long dry season, it still persists.

A further cause of failure to resist attack is negligent cultivation. It was, in Java, amongst the badly cared for gardens grown by natives under the "culture" system, that the disease first proved destructive. No exact work appears to have been done in India or elsewhere on the influence of manures, different methods of pruning, good cultivation and the like, on the incidence of rust attacks, but it appears to be an accepted maxim in certain parts of South India, that generous treatment of the bushes is essential if they are to face the attack successfully. The best planters may, indeed, sometimes be found to hold leaf disease in contempt; there is no doubt but that a marked improvement in the direction of reduced losses has occurred since the time of the first outbreak following 1870, and that this is largely due to better cultivation and, particularly, manuring; but there is reason to believe also that the virulence of the disease has lessened, and part of the improvement may be due to this.

There are many scattered records of the beneficial effects of heavy manuring in leaf disease ; thus, in Tonkin, an attack on bushes exhausted by over-production was checked by the application of about  $\frac{1}{2}$  cwt. of cattle manure per bush ; in the New Hebrides, after the first panic, which led to many estates being almost abandoned, a revival set in about 1911, when it was found that good cultivation and manuring greatly reduced the losses ; in Sumatra, green manuring carried out twice in rapid succession gave a new lease of life to bushes moribund from the disease. Such instances can be multiplied and go far towards establishing the value of heavy manuring in fighting the rust.

In the same manner, other conditions which tend permanently or temporarily to weaken the bushes, render them more liable to damage from this disease. A shallow or unsuitable soil is a predisposing cause. So also is over-production, which has been observed to break down the resistance of a relatively immune bush. But these are merely illustrations of the general principle that constitutional weakness often prevents successful resistance to parasite diseases.

From the early days of the disease, attempts have been made to find resistant varieties. Liberian coffee was at one time thought much of in this respect, and was certainly, on the whole, more resistant than arabica in many places, losing its leaves less readily under attack ; but it has failed to maintain its reputation, in Java at least, where it is now thought to be as much damaged as any variety. A good deal of hybridisation work has been done in South India and elsewhere, sometimes with temporary success, but, apart from the doubt whether the parents were inherently more resistant than the general level of the crop, the work was not, as a rule, scientifically controlled and any success in individual cases was usually swamped after a while through want of selection and isolation. The varieties of *Coffea arabica*, *C. liberica*, and the Asiatic species chiefly available for hybridisation in the last century are probably not markedly different in their power of resisting the disease.

From the beginning of the present century, however, a stream of new African forms, chiefly from the Congo and West Coast regions, have reached the East, and their cultivation, especially in Java, has given results which promise to revolutionise the position of the coffee industry in the countries afflicted with leaf disease. The chief of these are *Coffea robusta*, introduced in 1901, and at present by far the most important ; *C. canephora* and its variety *Kouilouensis*, the latter known as Kouilou or Quillou coffee, introduced about the same time but not



distributed on a large scale until later ; the " Uganda " coffee, probably also a variety of *canephora*, a less heavy yielder than the last two (to which it is botanically related) but offering a good field for selection : *C. excelsa*, introduced in 1905 and said to be more vigorous and less injured by rust than *liberica*, which it otherwise resembles ; *C. congensis* var. *Chalotii*, introduced in 1908 and said to be practically immune, but giving a weaker growth and lower yield than *robusta* and its allies, Quillou and Uganda ; *C. abeocuta* and *C. stenophylla*, which appear to be used chiefly for crossing, but are being tested on a larger scale in some places ; *C. Laurentii*, probably simply a form of *canephora*, recently spoken highly of, and a few others. None of these appear to be pure types, some, such as *robusta*, varying largely and requiring selection and isolation if the best results are to be expected ; but, so far, they are, as a class, less injured by *Hemileia* than the varieties formerly grown, and under the scientific control of the Java Agricultural Department may be still further improved. The rapidity with which they have found favour in Java may be judged by the area and export figures for *robusta* in recent years. In 1906, there were less than 10 acres of *robusta* grown, in 1913, more than 100,000. Over 24 million new plants are said to have been planted out in the four years, 1907 to 1911. In 1910, the export of *robusta* was about 1,900 tons as compared with 6,000 tons of *arabica* and 4,700 tons of *liberica*, in 1913, the estimated figures were 18,500 tons *robusta*, 6,975 tons *arabica*, and 3,360 tons *liberica*. The yield of this variety is heavy and it comes into bearing rapidly, but the quality is poor, possibly some of its hybrids with *liberica* or other varieties will prove more satisfactory in this respect, for they also appear to resist *Hemileia*. Disquieting statements have also been made that it suffers more from the disease now than when first introduced. Of the new varieties, only *C. excelsa* appears to yield a really good quality product, approaching *arabica*, but this form is still in the experimental stage and cannot yet be recommended. It is probable that no single type will succeed in ousting the others. Each has some character fitting it for special conditions of soil and climate. Thus *robusta* is said to have done very well under heavy rainfall, though requiring a well-drained soil as it is sensitive to water-logging ; Quillou (also a heavy yielder) has been found to do better than *robusta* on dry estates with heavy soils ;\*

\* Under this name, two distinct forms seem to be included, as the Quillou now widely grown in Java is said to differ definitely from the more recently imported specimens of *C. canephora* var. *Kouilouensis*.

some of the *liberica* allies, as *excelsa* and *abeocuta*, seem able to stand a heavy rainfall in heavy soil, and so on. How far their resistance to leaf disease will be maintained, remains to be seen: individual cases of damage have already been reported; but it must be remembered that some of the earlier introduced forms entered cultivation as anything but pure types; in the mixture there may have been susceptible races; and the steps now being taken to secure pure races may enable these less resistant forms to be eliminated.

In Madagascar, also, the newer forms seem to have done well. Kouilou coffee, introduced in 1900, was reported a few years ago to be next in importance to *liberica*, which is the variety chiefly cultivated. *Robusta* is also stated to be giving good results, while *C. congensis* var. *Chalotii*, introduced about 1902, is very highly spoken of by some authorities for the east coast of the Island, a region where *Hemileia* rapidly destroys *arabica* and seriously damages *liberica*.

Spraying against leaf disease has been tried in various countries in the past, but, in general, without satisfactory results. Recently, with better appliances and methods, it has been revived with success in East Africa and promising experiments have been made in South India. It is recommended to spray once with Bordeaux mixture, preferably in May. The under surface of the leaves, and also the stem, and fallen leaves on the ground, should be covered, and large blocks should be treated together so as to avoid re-infection. For this purpose battery sprayers (see p. 134) are probably the most suitable. The cost is somewhere about Rs. 15 per acre, which is not considered prohibitive on coffee, but it varies in different estates, chiefly according to accessibility of water and cost of labour. Where the December attack is usually the most severe, spraying in September is recommended. Resin-Bordeaux should be used for the May spraying, but the adhesive is not required in September.

At present it may be said that there is every prospect of successfully fighting leaf disease by the choice of suitable resistant varieties, coupled with good cultivation, and liberal manuring, while further experiments with regard to spraying are necessary.

Forms of *Hemileia vastatrix* have also been recorded on species of *Gardenia* in Java and China, but, as in the case of the similar form on *Canthium*, there is no proof that they are identical, at any rate to the extent of being able to infect coffee.

**Koleroga** (*Corticium Koleroga* (Cke.) v. Hoeh.-*Pellicularia Koleroga* Cke.).—This disease (which is sometimes known locally as black rot, the native name "Koleroga" being applied to more than one disease) has been known in Mysore about as long as the coffee leaf disease, but the nature of the parasite causing it has only recently been recognised. Various records exist of its occurrence in the West Indies, Porto Rico, Guatemala, Surinam, and Venezuela in the New World, and in Java, New Caledonia, and the Congo in the old. There is much doubt, however, in the majority of these cases, whether the fungus is the same as that first described from Mysore. Even the Venezuelan specimens, examined though they were by the same authority that named the Mysore fungus and pronounced to be identical, are now said to differ in important particulars. Within the last few years, the Surinam and Porto Rico diseases have been considered by one observer to be due to *Pellicularia Koleroga* and by another to an allied but quite distinct fungus. It seems probable that several different fungi attack coffee in a somewhat similar manner, and except for New Caledonia and Porto Rico, it is doubtful if any other country but India suffers from this particular disease. It has been little studied locally, and our knowledge of its pathological characters and the conditions under which it flourishes is far from complete.

In Mysore, the districts most affected by this disease are those towards the Western Ghats, where misty weather is frequent. In this region, it does much more damage than *Hemileia*. Further inland, where the mists are less, *Hemileia* is common and *koleroga* comparatively rare. Coorg, and the Nilgiri and Shevaroy Hills, are other localities affected.

The attack usually becomes evident about July. The leaves of affected bushes are found covered to a greater or less extent, on their under surfaces, with a fine, membranous film, greyish-white in colour, smooth, and somewhat brittle when dry, but flexible and easily peeled off when wet. This membrane may be traced in a narrow band down the petiole and along the branches, sometimes for a considerable distance, the general tendency



FIG. 192. *Koleroga* of coffee (*Corticium Koleroga*): affected shoot.

being to spread towards the tips of the branches. When wet, it is very transparent and hard to detect; when dry, on the contrary, it is shiny or glistening. The membrane frequently spreads over the berries, and, in severe cases, leaves, flowers, and fruit may be bound together by the web-like films. The older stems are usually free from it.

The affected leaves turn black and become detached from the twigs, but are usually held suspended by the bands uniting the fungus on leaf and stem. The bushes may easily be recognised by the hanging masses of blackened leaves, which, in severe cases, may involve the greater part of the foliage and fruit on the bush. In wet weather, these decompose quickly and a brownish liquid may drip from them. The effects are therefore much more intense, so far as rapid destruction of the green parts is concerned, than with *Hemileia*, but fortunately require a higher humidity for their full development than is usually found. In this respect, it appears to be more restricted in range than thread blight in tea, with which it is otherwise closely allied.

Under the microscope, the film is found to be composed of a closely interwoven web of hyaline, septate hyphae, 5 to  $7.5\mu$  in diameter, much branched, and crisscrossing in all directions. The web is capable of great extension laterally, often covering the whole under surface of the leaf, but is usually only one or a few layers in depth, condensation into thick strands, such as is sometimes found in the allied thread blight of tea, being very rare or absent. In the New Caledonia form, certain hyphae divide by repeated dichotomous branching at short intervals to form a sort of disk; these disks are said to fix the film to the leaf, and also to act as suckers, as it has been found that the cuticle of the leaf under them is corroded to such an extent that on removing the film the position of the hyphae can be traced by the hollows eaten in the cuticle. These hollows never actually penetrate into the epidermal cells.

At certain points, particularly towards the end of the hyphae, small roundish swellings are found laterally, attached to the filament, when immature, by a short stalk, but almost or quite sessile when fully grown. They are hyaline, and provided with small roughenings or warts on the surface, the whole body being about  $7.5\mu$  (or  $10\mu$  in New Caledonia) in diameter. These were formerly taken for spores, though attempts to germinate them failed; it is now said that the bodies seen by earlier observers were the basidia, which were mistaken for spores. These basidia arise in groups of from 2 to 5 at the end of short lateral branches. Each basidium is egg-shaped, 11 by  $8\mu$  in diameter, and bears 2 to 4 hyaline, thin-walled spores, 10 to 12 by  $4\mu$  in diameter, on short sterigmata.

The fungus appears to be a pure ectophyte, after the manner of thread blight on tea; at least no haustoria have been made out, and the film appears to be quite superficial. Nevertheless the injury caused is intense, the affected leaves and fruits turning black and being entirely killed. The action appears to be too severe to be accounted for by the mere blocking up of the mouths of the stomata, as the earlier observers believed, and it is more probable that there is a direct absorption of nutrition by osmosis from the leaf.

Spread appears to be partly, as in thread blight, by the blowing about of infected leaves bearing living mycelium, partly through spores.

Good results have been obtained by spraying with Bordeaux mixture in South India. The disease is easier to control in this manner than leaf disease, since it is usually restricted to definite areas in an estate. Spraying should be done in May, and where the monsoon attack of leaf disease occurs, one spraying will do for both. It is naturally advisable also, where possible, to remove foci of infection by cutting out and burning attacked branches. Sometimes good results have been obtained by altering the wind belts and promoting currents of air in stagnant localities.

The fungus has been observed on several plants besides coffee, including *Strobilanthes*, dahlias, and crotons.

**Pink disease** (*Corticium salmonicolor* B. & Br.). Coffee is amongst the plants which suffer most severely from this widely distributed parasite. There is, as yet, little information regarding it on this host in India, except that it is known to occur, but, as it appears to be a serious disease in the Malay Peninsula and Java, and as the fungus is common on rubber and other plants in South India, it is probable that Indian coffee bushes do not escape its ravages. It is described under rubber, on p. 499 below. In coffee, it is not uncommonly found on the fruit.

**Stump rot and other root diseases.** Coffee is not less subject to this class of disease than tea, and at least two (and probably more) varieties of root disease occur in Mysore. It is quite common to find patches consisting of from one or two, up to 150, bushes killed by some form of root disease in Mysore and Coorg, and in one extreme case, an area of some 40 acres was wiped out in about 18 years and all attempts at replanting failed. In this particular case, the attack, when seen about 12 years ago, was still progressing, recent cases being numerous in the bushes at the edge of the patch.

Perhaps the commonest form in Mysore is that in which small patches, consisting of one or more dead bushes, occur scattered throughout an estate. In every case they can be traced to the presence of the stump of some cut-down jungle or shade tree. Old planters state that they have known of two distinct outbreaks in certain estates, the first commencing a few years after the jungle was cleared in opening the estate and lasting many years, and the second after removing the shade trees first made use of. In rare cases, the attack may not commence for as many as 20 years after the tree has been felled. By digging out

the whole of the root system of the infected bushes, or by isolating them by a trench from the healthy plants, spread may be checked and the loss remains slight. Even where no treatment is attempted, the spread, which is fairly rapid to the first few bushes, gradually slows down and ultimately ceases.

On digging up the dead bushes, the tap root and larger lateral roots will be found encrusted with a gritty mass of earth, small stones, and fungus hyphæ, brownish in colour, and almost of the consistency of charcoal. If this be scraped off, the wood below is found marked by brownish or almost black areas, sometimes with a paler centre but always bounded by a sharp black line. Though no fructification has as yet been found, these characters indicate with practical certainty that the cause of the disease is identical with that more fully described above (p. 429) as causing "brown root disease" of tea due probably to *Hymenochaete* *noxia*. It has been observed in Mysore to spread to various species of *Ficus* and to *Grevillea robusta*, the characters of the rot produced in the deeper layers of the root differing somewhat in each case.

The following list of trees said to be capable of starting stump rot of some kind or other in South India may be of interest, though it is by no means complete, nor believed to be entirely reliable. The local vernacular names are in brackets :—

<i>Acrocarpus fraxinifolius</i> ( <i>howlge</i> )	<i>Ficus infectoria</i> ( <i>basuri</i> )
<i>Artocarpus integrifolia</i> ( <i>jak</i> or <i>halasa</i> )	.. <i>Tsiela</i> ( <i>bih basuri</i> )
<i>Cinnamomum iners</i> ( <i>yellage</i> )	<i>Garuga pinnata</i> ( <i>gwoddan</i> )
.. <i>zeylanicum</i>	<i>Myrsine magnifica</i> ( <i>ramanadike</i> )
<i>Dalbergia latifolia</i> ( <i>biti</i> )	<i>Soyimida febrifuga</i> ( <i>kul garige</i> )
<i>Eriobotrya japonica</i> ( <i>loquat</i> )	<i>Sponia Wightii</i> ( <i>gurcul</i> )
<i>Ficus glomerata</i> ( <i>atti</i> )	<i>Terminalia belerica</i> ( <i>tari</i> ).

Some of these are probably themselves victims, like the coffee, but not necessarily capable of starting the disease. *Grevillea robusta* is liable to attack, but many think it does not cause stump rot in coffee.

The description above on p. 429, can be referred to for further details, and for treatment. Coffee bushes have been successfully replanted in the diseased patches a year after removing the affected bushes.

A more severe form of root disease has been seen in the part of Mysore towards the Ghats, where the rainfall is very high. In this disease, not only does extension continue to occur for many years, but the soil remains infected to such an extent that replanting has been tried without success. It is probable that still other root diseases occur in the coffee districts, but they have not been investigated. In Ceylon,

a *Rosellinia* is said to have probably assisted in the extermination of coffee; and in Java, several different types of root disease have been described. In no case, however, has the cause of the disease been definitely ascertained, and no suggestions can be made for treatment beyond trenching in the early stages and uprooting and burning the affected bushes with a view to preventing spread. Stumps, whether from jungle or shade, should, of course, be uprooted if belonging to trees liable to start disease. The use of a Trehwella Jack greatly facilitates this work, but should it be impossible for any reason, the stump should be isolated by a trench at least three feet deep.

**Leaf spot** (*Mycosphaerella coffeicola* Cke.)—Coffee leaves bearing this fungus have been collected in the Wynaad. It seems to be rare in the East, having been previously recorded only in the New World.

The leaves are marked by small, roundish spots of a greyish-white

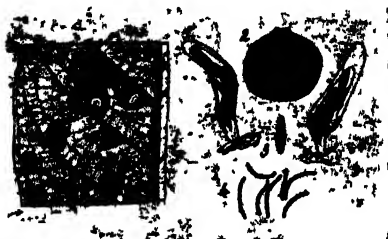


FIG. 193. Coffee leaf spot (*Mycosphaerella coffeicola*)  
1, spots on leaf,  $\times 4$ ; 2, perithecial surface view,  $\times 100$ ; 3, asci with ascospores,  $\times 200$ ; 4, pycnospores,  $\times 200$ .

colour, surrounded by a narrow, deep brown band. Further out, there is a diffused zone of a paler brown, which gradually merges into the green of the healthy tissues. On examining with a lens, minute, black points can be seen, chiefly on the upper surface.

The black points are fructifications which, in the Indian specimens as in those first described from Venezuela, consist of two forms, a perithecial and a pycnidial.

The perithecia (Fig. 193, 2), are immersed in the tissues, only the mouth breaking through the epidermis. They contain club shaped, curved asci, 50 to 60 by 15 to 20  $\mu$  in diameter, with 8 spores, and without paraphyses (Fig 193, 3). The spores are biserial or crowded and up to 25 by 4.5  $\mu$  in diameter, though those from the Wynaad, which were probably immature, were only 18 by 2.5 to 3  $\mu$ . They are faintly yellow in colour, fusiform, 1-septate, constricted at the septum, and with one cell often longer and more pointed than the other.

The pycnidia are of the type characteristic of the imperfect genus *Septocia*, and contain filiform, hyaline spores, up to 44  $\mu$  long by 1.5  $\mu$  broad, curved, and with several

septa (Fig. 193, 4). Those found on the original Venezuelan material (*Septoria maculosa* (Berk.) Cks.) are described as being  $20\mu$  long and with about 7 nuclei.

Whether these two spore forms are stages in the one fungus is not known.

The damage done by this disease is trifling, so far as is known.

**Brown blight or anthracnose** (*Colletotrichum coffeanum* Noack = (?) *Glomerella cingulata* (Stonem.) S. & v.S.).—This fungus, which is believed by recent workers in the United States to be identical with the widely distributed *Glomerella cingulata*, is fairly common in South India (Coorg, the Wynnad, the Nilgiris, etc.), usually on the leaves, but at times also attacking the berries. It is known in Brazil, Costa Rica, Porto Rico, East Africa, and probably also in Java, Madagascar, and Réunion, though the name usually given it in the last three instances is *Glœosporium coffeanum* Del., and it is not certain that it is the same species as the American one.

In many cases the fungus is associated with Hemileia, but it also occurs alone, and in the attacks on the twigs and berries seen in India there was no trace of the rust fungus.

On the leaves, the spots are roundish or irregular, those at the margin being often elongated, and the main nerves of the leaf limiting the spread in certain directions. The largest spots may be nearly an inch in diameter. The colour is at first brown, then grey, and the edge not as distinctly bounded by a darker band as in the *Mycosphærella* spots. The spore acervuli are visible as small, black dots, mostly on the upper surface of the leaf.

The hyphæ penetrate the tissues with a less limited growth than in *Mycosphærella*. They seem not to enter the cells, but only to pass between them, killing them by some process of diffusion or absorption. Stromata are formed by a collection of the hyphæ under the cuticle, chiefly on the upper surface of the leaf, rather evenly scattered over the spot. They consist of two or three layers of pseudoparenchyma, the cell walls being brown. At the surface is a layer of erect, almost colourless spore stalks, cylindrical in shape, and 18 to 20 by  $4\mu$  in diameter. Each bears a single conidium. Other cells of the outer layer grow out into dark brown, stiff bristles (setæ), some four or five times as long as the spores, unseptate or with one or two cross walls, and pointed at the tip. These setæ may be absent and are especially rare in young acervuli, and in such cases the fungus may be classed as a *Glœosporium*. As previously mentioned on more than one occasion, the distinction between these two genera is artificial and should not be maintained.

The conidia are irregularly cylindrical, rounded at the ends, often somewhat curved or indented, hyaline singly but pale pink in mass, and measure 12 to 18 by 4 to  $5\mu$  in diameter. As they develop, they rupture the cuticle of the leaf and appear on the surface.

On the berries, the spots are more limited and the bristles well developed. The effect is to form a depression or to check the growth of the berry on one side. At this point, a black or brown stain is found on the



bean after pulping, and such beans have to be picked out as they lower the quality of the produce. Fortunately this form of attack appears to be uncommon, though a serious outbreak has been reported from Coorg, and it is spreading in Mysore.

In Brazil, the twigs are sometimes attacked, the fungus producing elongated, sharply defined spots, surrounded by a slight marginal line. In Uganda, the fungus also attacks the twigs and does considerable damage, being at least one of the causes of the destructive condition known as "die back." The affected plants lose their leaves, and the branches wither and dry up. It usually begins at the tip and works back, but may start in the middle and spread both up and down. In India, a closely allied fungus was found associated with a twig disease, which caused some alarm in the Nilgiri and Nelliampati Hills about a dozen years ago and has spread considerably since. This species was without bristles and therefore a "Glœosporium," but not on that account, necessarily different from the present fungus. The acervuli were also considerably smaller than those found on the leaf and fruit, and the spores a little shorter. Whether these differences are enough to separate the two fungi is a question which cannot be decided without further study.

The Nilgiri twig disease (Fig. 26), was reported in 1904 to be spreading on all high-elevation coffee estates on the Nilgiris, and was considered by some experienced planters a worse enemy than Hemileia.

The attack begins near the tips of the twigs and usually spreads backwards to a limited extent only. In full-grown bushes, it commences on the primary branches here and there indiscriminately throughout the bush, killing off the secondary twigs on each attacked branch, except those towards the base, but not usually killing the entire branch. In

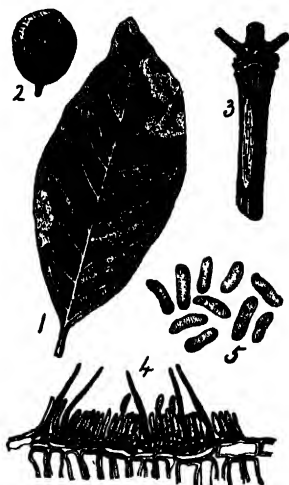


FIG. 194. Brown blight of coffee (*Colletotrichum coffeanum*): 1, attack on leaf,  $\times \frac{1}{2}$ ; 2, attack on berry, nat. size; 3, attack on twig, nat. size; 4, section of acervulus showing conidia and setae,  $\times 230$ ; 5, conidia,  $\times 460$ . ( $\frac{1}{2}$  & after Noack).

young bushes, on the other hand, it commences on the lower primary branches, and as the bush increases in size, the lower primaries die off. When the disease was at its height, it became quite difficult to raise a healthy young plant.

The attacked branches are covered with corky bark higher up and more irregularly than usual. Their leaves early fall, and soon new shoots are put out from the leaf axils to replace the lost foliage. These bear small leaves, which are often pale and crumpled; eventually they die back in turn. The leaves and berries are sometimes directly infected, the former showing brown patches along the margins, while the berries are marked by deep purple, sunken spots. The effect on the crop is most destructive. The berries on the attacked branches generally drop off, those remaining being rarely larger than a pea. The loss in affected blocks sometimes exceeds half the crop.

It was not proved that the *Glœosporium* was the cause of the disease, but no other parasite could be found, and the disease itself bore a distinct resemblance to the "die back" of branches known to be caused by related species, as, for instance, in citrus fruits. The acervuli were found only on the twigs, from just beyond the limit between the dead and healthy tissue to the end of the twig, and the mycelium was traced into the wood, not only in the dead portions, but back to parts externally sound.

Spraying with Bordeaux mixture was found of value in checking severe attacks of this twig disease and would, no doubt, be equally useful against the leaf and fruit forms of *Colletotrichum coffeanum*. About two months after the first spraying, the new shoots that had been put out were reported to have a fairly healthy look and to appear free from the fungus. A second spraying was then given, and three months later it was reported that two-thirds of the bushes threw out strong, healthy wood with only here and there a sickly twig. The remaining third had several diseased twigs. These were heavily pruned, as many sickly branches as possible being removed and burnt. About 18 months later the block sprayed was reported to have remained "not quite, but almost, free from twig disease."

The disease did not appear to spread readily, except where the bushes were contiguous. Isolated blocks escaped infection at least for a time. This is one of the few cases seen in India where the provision of "wind breaks" (see p. 109), to check dissemination of spores through the air, might have been advantageous.

An allied species, *Colletotrichum incarnatum* Zimm., has been found in Java, not only on leaves affected by Hemileia but also on leaves and stems unassociated with the rust. Especially is it frequently found on dead stems, but whether it causes their death, or is secondary, remains doubtful. It has also been found in German East Africa on leaf spots caused by Hemileia. This species does not, as described, differ in any important particular from *Colletotrichum coffeanum* and is very possibly identical.

**Brown eye spot** (*Cercospora coffeicola* B. & Cke.).—This is one of the most widely distributed diseases of coffee, having been reported from most coffee-growing parts of the New World, from East Africa and Uganda, from Java, and from New Caledonia. In India, it has been found in Mysore.

The fungus occurs on the leaves and berries, causing small, circular spots on the leaves and larger blotches on the berries. In Java, it has been seen also on the green twigs. Usually the damage from the leaf spot is small, but it has been reported to have seriously injured, and even killed, nursery plants in Uganda. On older plants, the berry attack (which has not yet been recorded in India) may do much harm by causing the pulp to become hard so that it adheres to the bean, and sometimes by destroying the latter.

The leaf spots are more or less circular when single, and one-eighth to one-half inch across. Each is at first bright brown, then the centre becomes greyish-white and a brown or reddish-brown ring defines the margin. This is most distinct on the upper surface. On the berry, the spots are most prominent on the upper side and may cover half the surface. The affected part turns black and shrinks, and the berry may fall before it ripens. Up to 70 per cent. injured berries have been found in the crop in places in Porto Rico, and the quality of the produce was somewhat reduced by these attacks.

The fructifications of the fungus are found on both sides of the leaf spot. Tufts of short, erect, unbranched conidiophores emerge from the stomata, each being from 50 to

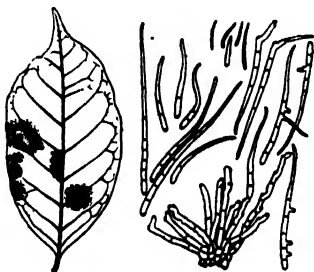


FIG. 195. Brown eye spot of coffee (*Cercospora coffeicola*): leaf with spots,  $\times \frac{1}{2}$ ; conidiophores and conidia,  $\times 200$ . The conidia on right have formed secondary conidia. Above, a group of secondary conidia.

150 $\mu$  long by 4 to 5 $\mu$  broad, with several septa, and the usual knee-bends near the top. The conidia are about the same length, narrowed above, straight or curved, colourless and with several (up to 7) septa. On germination, they may either give out long hyphæ, or bear secondary conidia, on small conidiophores, from the base or side of the spore. The secondary conidia are much smaller than the primary, often unseptate, and have not been seen to germinate.

In Java, robusta coffee is said to be more readily attacked than arabica or liberica. In Uganda, only the berry attack has been seen on robusta. In Surinam, robusta, abecuta, stenophylla, and arabica are attacked.

In bad cases, spraying with Bordeaux mixture is recommended.

**"Cobweb" disease** (sterile mycelium).--Under this name, a disease has been described in Java, which occurs also in old coffee plantations in Sylhet and is probably present in other parts of India. It bears a certain resemblance to koleroga, and has been sometimes taken for the latter.

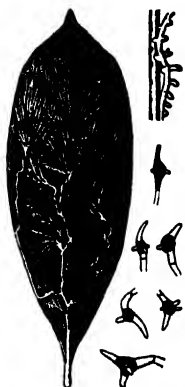


FIG. 196. "Cobweb" disease of coffee: affected leaf, under surface,  $\times \frac{1}{2}$ ; hyphæ of the mycelium, and anchor cells (after Zimmermann),  $\times 500$ .

The fungus is found on the twigs and leaves, forming thick, branching, white strands, which spread out on the under surface of the leaf into a fine, cobweb-like layer. This can be peeled off when moist, just as in koleroga. The affected leaves lose their green colour, become flaccid, and fall. Extension occurs to other leaves and twigs in contact, and a large part of the bush may be destroyed, the twigs dying as a result of loss of leaf but not seeming to be directly injured by the fungus. As in koleroga, only moist localities are affected, and heavy shade favours the disease.

The mycelium seems to be purely superficial, extending as bands of long, straight, parallel hyphæ, from which a finer network spreads over the leaf surface. The hyphæ are of two kinds on the leaf: a larger, straight, relatively little branched, next the epidermis, and a finer, curved, much branched, outside. The branches from the latter are short, and would suggest fixing or feeding organs, except that they are not on the inner side of the fungal layer.

No reproductive stage has been observed, the only specialised bodies found being peculiarly branched, thick-walled cells, to which the name "anchor cells" has been given. Each is formed as an elongated, somewhat spindle-shaped cell on the end of a hypha, and, when mature, the yellowish contents are collected in the middle, surrounded

by a thick, hyaline wall, from which several processes arise. There is usually one long process at each end, and one to three laterally. Their function is unknown.

The treatment is the same as for *koleroga*.

**Sooty mould** (*Capnodium brasiliense* Puttem.).—As is general with the sooty moulds, this fungus is not a parasite and does not feed on the bush itself, but on the sugary "honey dew" found on leaves and twigs infested with certain insects.

The cases seen in South India were associated with *Lecanium hemisphaericum*, *L. viride*, *Hemilecanium imbricans*, and *Pulvinaria psidi*, as well as aphids. In Ceylon, an allied (perhaps identical) fungus has long been known, associated with *Lecanium nigrum* and allied insects, to such an extent that an early observer says "whole estates are seen black with bug, that is, with the fungus." Species of *Aphis*, *Dactylopius*, and *Pulvinaria* are known to induce sooty mould in coffee in many countries.

As it occurs in South India, the fungus is characterised by its extraordinary diversity of spore forms, and extreme reproductive activity. No less than four different forms of conidia, besides a pyrenidial, and an ascigerous stage, are found on the same mycelium.



FIG. 197. Sooty mould of coffee (*Capnodium brasiliense*): 1, leaf with sooty mould,  $\times 4$ ; 2, mycelium; 3, *Brachysporium* conidia,  $\times 330$ ; 4, *Coniothecium* conidia,  $\times 330$ ; 5, *Triposporium* conidia,  $\times 330$ ; 6, group of pyrenidia and perithecia,  $\times 130$ ; 7, tip of pyrenidium with pycnosporores,  $\times 660$ ; 8, asexual spores,  $\times 330$ ; 9, ascospores,  $\times 660$ . (29 after Puttemans and Maublanc).

The mycelium forms a more or less dense, black crust on the leaves and twigs of bug-infested plants. This has at first the appearance of a thin, black wash, chiefly on the upper surface of the leaves, but later increases in density and becomes granular and even woolly where the growth is most vigorous. It may last as long as 12 months and then dries up and peels off in large flakes, usually, if the bug persists, to be replaced by a fresh growth.

The hyphae are greenish-black, composed of unequal cells, constricted at the septum, and 6 to  $10\mu$  in diameter, or more regularly cylindrical and about  $3\mu$  thick (Fig. 197, 2). Certain hyphae break up into chains of almost cubical cells, somewhat convex on the lateral faces and 4 to  $6\mu$  in diameter. This is the conidial form corresponding to the imperfect genus *Torula*. In other places, collections of roundish cells, formed by the division of a hyphal cell in all three dimensions, so as to result in a compact mass, occur, and form what is known as the Coniothecium stage (Fig. 197, 4). On other hyphae, elongated, narrow conidia measuring 40 to 60 by 3 to  $4\mu$ , greenish-brown in colour, broader at the base than above, and with 5 or 6 septa, are borne laterally at the end of short stalks. This form is known as *Brachysporium* (Fig. 197, 3). Other spores similarly borne are formed of 3 or 4 arms united in the centre in the manner of a star-fish, each arm composed of 4 or 5 cells; the whole body becoming readily detached and germinating as a single spore (Fig. 197, 5). This "*Tripsoporium*" stage was one of the first sooty moulds described, in connection with coffee bug in Ceylon, but it is not certain that the Ceylon and Indian species are identical, as several forms of sooty mould are known to occur on coffee in different parts of the world. All the above-mentioned conidial forms can germinate and reproduce the fungus.

A pycnidial form is found on most affected leaves and twigs, but is by no means invariably present. It consists of elongated, spindle-shaped or almost cylindrical bodies, usually unbranched but sometimes forked, up to  $250\mu$  long by  $40\mu$  broad, open at the tip, and containing large numbers of small, hyaline, elliptical spores, 5 by  $2\mu$  in diameter (Fig. 197, 6-7).

The perfect stage consists of small perithecia, from 35 to  $60\mu$  broad, sometimes on a thick stalk, 30 to  $40\mu$  in diameter, or occasionally formed as swellings laterally on a pycnidium. The top is open and fringed with small teeth. Inside are a number of asci of variable shape, narrow below and swollen near the top or in the middle, and 30 to 35 by 11 to  $26\mu$  in diameter (Fig. 197, 8). Each ascus contains 4 to 8 aeciospores, at first hyaline then dark smoky green, 3-septate, the second cell from the top broader than the rest, and 13 to 16 by  $5\mu$  in diameter (Fig. 197, 9).

The growth of this fungus is closely associated with the honey-dew induced by the insects with which it occurs. The liquid is formed in such quantities as often to drop from the bushes, carrying spores of the mould, which continues to propagate in the undergrowth so long as it has sufficient of the sugary juice to feed on.

Though exceedingly unsightly, the damage caused by this growth on the bushes is much less than one would expect at first sight. This is because it is not parasitic, feeding so far as is known only on the honey-dew. Its chief powers for harm lie in the fact that it cuts off an appreciable proportion of the sun's rays falling on the leaves, and so interferes with the activity of the leaf in manufacturing food. To a lesser extent, it may block the stomata in some cases, but as it is chiefly on the upper surface where stomata are rare, this cannot often occur.

The treatment is obviously to deal with the insect responsible for the production of honey-dew. As soon as the activities of the insect lessen, the fungus disappears rapidly. A bush cleared or nearly cleared of the insects mentioned will never be found to maintain a growth of

sooty mould. Fortunately most coffee planters are now alive to the necessity for fighting the various coffee bugs and scales, and " estates black with fungus " are not likely to be met with commonly now-a-days. In many cases, the chief seat of the insects is in the shade trees, and a favourite remedy is to remove the shade. Only certain trees are liable, and their use is being discontinued where scale is common.

## CHAPTER XIV.

### RUBBER.

(*Hevea*, *Manihot*, *Castilloa*, *Ficus*).

As with coffee, our knowledge of the cryptogamic diseases of rubber-producing plants is largely based on work done in other countries; the local peculiarities of these diseases, their distribution, intensity, and the influence on them of climatic and other external conditions, which are oftentimes so important in devising methods for their control, have been little investigated in India. Valuable work has, however, been done in regard to "pink disease" and "black thread," which at present seem to be the most serious diseases of the Para plant in India.

**Pod rot and canker** (*Phytophthora Faberi* Maub.).—This fungus is best known as the cause of the formidable canker and pod rot of cacao, found in almost all cacao-growing countries. The *Hevea* attacks are quite similar; and in Ceylon and Java, *Hevea* canker has been artificially produced by inoculating with the fungus from cacao. Experiments with the latter fungus have further shown that it can attack seedlings of various garden flowers, as well as young tomato and brinjal plants; while in nature it has been found also on breadfruit and papaya.

The disease has been reported from South India, the Philippines, Java, Samoa, New Guinea, the Cameroons, the West Indies, and Surinam, chiefly on cacao.

On the fruits, it is not certain how many of the cases described are due to this fungus, and how many to that which causes "black thread" and is described below. A rot is produced, characterised by a sodden, watery discoloration of the pods while still attached to the tree. The fruits then turn black, and the outer soft layer ultimately shrivels and splits, but the woody wall below does not open to liberate the seeds. From the fruits, the rot may pass back through the stalks to the green branches and kill these for a short distance, but no case is known in which it has reached the main stem and set up canker. The stem canker, when it occurs, is said to be always the result of an independent local infection.



Apart from the loss of seed (a minor matter now-a-days), and the possible risk of spores from the fruit setting up stem canker, the fruit rot is of little consequence.

Canker of *Hevea* stems has only proved to be a serious disease, in Ceylon, in those cases where the rubber was interplanted on old cacao estates. It is suggested that this is due to continued infection of the rubber from the cacao, which, when old, usually provides an abundant supply of infective material. Pure rubber, and rubber and tea estates have suffered little. In Java, canker is more prevalent on old than on young trees, 55 per cent. having been attacked in one case reported.

The symptoms of *Hevea* canker are sometimes inconspicuous on external examination. On young trees, the bark may appear darker than normal, but this is not visible once the thick, brown bark develops. In some cases, the bark exudes a reddish or purplish liquid, which may be noticed in very wet weather, even when the patch of disease is small,

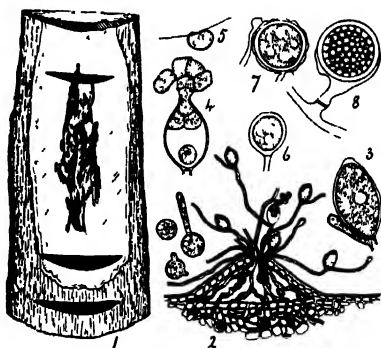


FIG. 193. Canker and pod rot of rubber (*Phytophthora Faberi*): 1, cankered stem with bark cut away to expose the discoloured bast, and deeper incisions above and below showing extension in the inner layers; 2, the fungus fructifying on cacao pod; 3, a sporangium with sub-sporangial branch; 4, sporangium discharging; 5, a zoospore; 6, a chlamydospore,  $\times 375$ ; 7, zoospore with antheridium; 8, parthenogenetic zoospore,  $\times 425$ . (After Rutgers from *Hevea*, 5-5 and 7 after Rorer from cacao, 8 after Coleman from cacao).

but usually only happens when a large area is involved. No latex is produced in the diseased area, and the stoppage of the flow in one or several cuts is often the first symptom of the disease.

On scraping away the bark of an affected patch, the underlying tissues are found discoloured. The part outside the laticiferous tissue is blackened, while the inner bark turns at first grey, with a distinct black border, and, later on, claret-coloured. The discoloured area is often much more extensive in the inner than in the outer layers (Fig. 198, 1). Frequently the bark is dirty red when cut, but darkens to purple-red soon after exposure. In the laticiferous vessels, the latex becomes coagulated, probably by substances produced during the growth of the fungus. Unlike the majority of cankers, the bark in this disease is frequently not cracked, and open wounds do not result, at any rate in the early stages. Severe attacks may, however, destroy the whole bark down to the cambium, over a large patch, and the dead tissue in these cases is ultimately cast off, leaving an open wound exposing the wood. Even in the milder forms, the bark may be much damaged by woody tumours and these may persist long after the disease has ceased to be active. It seems probable that many cases in which the bark is deformed and cracked, or contains woody nodules or "burrs," are old canker cases.

The mycelium of the parasite is composed of non-septate hyphae of irregular diameter, in places narrow, in others quite thick. They run at first between the cells of the affected tissues, but later on are said to penetrate the cells. After the internal growth has progressed for a time, branches emerge to the surface. On cacao pods, these arise from condensations of mycelium below the epidermis (often in the air spaces below the stomata) which, by their pressure, rupture the epidermis (Fig. 198, 2). They give a superficial growth, at first consisting for the most part of long, unbranched sporophores, which early become decumbent and form, on cacao, a rather dense hyphal network on the surface. On Hevea the surface growth is usually scanty.

Sporangia are formed at the ends of the sporophores, but when about half grown, the stalk continues its growth from immediately below the sporangium, sometimes pushing the latter over to one side, as in *Phytophthora infestans*, but without the swelling of the conidiophore below the insertion of the sporangium characteristic of the latter species (Fig. 198, 3). Sometimes the sporangia are borne on short lateral branches. They are egg-shaped or lemon-shaped, hyaline, with a prominent papilla at the tip, and measure 30 to 60 by 21 to 30  $\mu$  in diameter; exceptionally large ones are said to reach 80 by 42  $\mu$ . On germination, they may either give out from 15 to 30 zoospores, of the usual type found in *Phytophthora* (Fig. 198, 4-5), or one or more germ-tubes on which, in turn, secondary sporangia may be borne.

Chlamydospores, similar to those described in *Phytophthora Colocasiae* and *P. parasitica*, are also formed, both in the surface growth on cacao pods and in pure cultures from cacao. They have been seen on diseased Hevea fruit from Burma (Fig. 198, 6) but in this case may have belonged to the "black thread" parasite. They are spherical, yellowish when old, 22 to 50  $\mu$  in diameter, and with walls 2  $\mu$  thick. They germinate, so far as observed, always by germ-tubes.

Oospores are formed in the surface growth on cacao (? and Hevea) pods, and also within the tissues of the cacao pod. Most observers have failed to see antheridia and, in some cases, have possibly mistaken the chlamydospores for oospores, but in Trinidad

true oospores, with a separate antheridium applied laterally (and therefore resembling the type described in *Sclerospora* or *Peronospora*, and not that of *Phytophthora infestans* and its allies) have been seen (Fig. 198, 7). Usually, however, no antheridium is formed and the oospores develop parthenogenetically (Fig. 198, 8). They are spherical, 33 to 40 $\mu$  in diameter, with a colourless, thick, smooth wall, and granular contents. Their germination has not been observed.

Infection occurs in cacao pods by the germ-tubes, either from zoospores, or from conidia that have germinated directly (and probably also from chlamydospores), penetrating the epidermis either across the cells or through a stoma. Once within the pod, growth is very rapid. In cacao, where the pods arise directly from the stem, extension back along the inside of the peduncle to the stem, and subsequent development of a stem canker at this point, is common; in *Hevea*, a similar extension to the green shoots on which the fruit is borne occurs, but cankers on the main stem usually result from independent infection by spores blown from the fruit or from diseased cacao near by.

Measures for the control of this parasite have been chiefly tested on cacao. As the fruit rot in this crop causes very serious losses, spraying has been undertaken primarily with a view to checking this form of the disease. At the same time, it is evident that, since much of the stem canker arises through infection from the fruit, either by means of spores blown from the fruit to the stem or by direct infection through the fruit stalk, a reduction in the amount of fruit rot should materially help to diminish the canker. Spraying with Bordeaux mixture has, therefore, been undertaken with good results in Trinidad, and as the practical details are more fully worked out seems likely to prove generally applicable where cacao suffers seriously from this disease. To check canker the excision of the cankered bark has long been practised with fair success in Ceylon. Suspicious patches on the bark are lightly scraped and if the tissue beneath is found to be discoloured, it is cut out. The wounds are not tarred or protected in any way and remain as long patches of permanently exposed wood, which gradually dries up. To avoid the consequent injury to the tree, the application of tar or some other preservative has been recommended. Further measures advised in Ceylon are the collection of diseased pods at frequent intervals and their destruction by burning or burying, and the reduction of shade.

In the case of *Hevea*, conditions are different and these recommendations require to be modified. The fruit is of little value, whereas the stem canker is a serious matter, owing to the stoppage of the latex flow at and near the diseased patch. Spraying of the fruits is hardly

practicable on plants in bearing and gives no direct return for the cost, merely lessening the risk of canker appearing. The use of Bordeaux mixture or any copper compound is said to be dangerous once tapping has commenced, as if any gets into the latex it causes "tackiness." Hence it is only safe on young trees. Excision of the diseased tissue is, therefore, the recognised treatment for Hevea canker. Once the cankered spot is detected (by no means an easy matter), all the discoloured tissue should be cut out and burnt. Stoppage of the latex flow should be a danger signal leading to careful search for canker. After removing the tissue, it is recommended, in Ceylon, to apply a mixture of cowdung and clay to the wound, to promote healing, if the wound be small. Large surfaces cannot be expected to heal and should be tarred or otherwise protected.

At the same time, since we cannot keep on cutting out cankers indefinitely without ruining the yield of latex, the prevention of infection may become of vital importance where new cases frequently appear. In such circumstances, it may be necessary to apply a fungicide to the stems, so as to destroy spores lodging on the bark. So far as is known, no definite experiments have been carried out to test the efficacy of this measure, or what fungicide can safely be used; and the comparative rarity of Hevea canker in pure rubber or rubber and tea estates, as opposed to those where the rubber is mixed with cankered cacao as occurs sometimes in Ceylon, may indicate that stem treatment with fungicides is not called for in India.

**Black thread and leaf fall** (*Phytophthora Meadii* McRae).—The name "black thread" is used in Burma for a serious disease of the tapped surface of Hevea stems, marked by the appearance of vertical, slightly depressed, black lines. It is also known in Ceylon, Malaya, Java, and South India. In Ceylon, Burma, and South India an abnormal second leaf fall in Hevea, associated with dying back of the branches, has been observed and seems to be due to the same fungus. Cacao is not subject to attack, but artificial inoculations have succeeded on leaves of ceara trees and seedlings of castor and of some garden flowers.

The dark lines of black thread extend in through the exposed bast and may penetrate the cambium and reach the wood. The blackening spreads laterally also, and may eventually cover the whole surface of the cut area. Vertical cracks and deeply sunken areas, from which white latex exudes, result. Where renewal of the bark is taking place from the

margin of the cut, a thick pad of coagulated latex may collect, causing the bark to bulge and ultimately decay, large gaping wounds—true cankers extending down to the wood—may eventually be formed. The tree is not killed, but the blackened parts soon cease to yield latex and the death of the cambium seriously interferes with the smooth and even regeneration of the bark, on which successful tapping depends. The cankers heal irregularly and with roughened ‘wound tissue’ (callus) and, as the disease extends, it becomes more and more difficult at each round to find bark suitable for tapping.

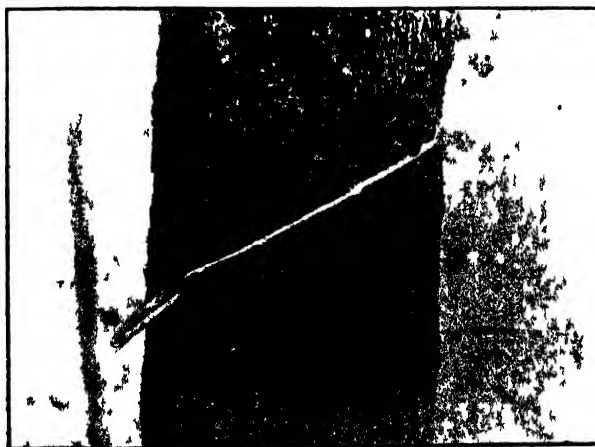


FIG 199 Black thread of rubber (*Phytophthora Meadii*) diseased tapping wound

The damage caused by this disease in Burma is increasing. On one estate where it was first noticed in 1913 the number of trees affected was 12 000 out of 77 000 in tapping by 1914 and 42 000 out of 117,000 in tapping by 1915. In 1914 the loss in rubber was estimated at 2 000 to 3 000 lb in 1915 between 8 000 and 9,000 lb.

The fruits are also affected and those attacked can easily be recognised by an exudation of latex in drops. The diseased areas are dull green and water soaked at first, then darken. Latex may collect between the soft rind and the woody shell of the seed capsule. The fruits remain hanging on the tree and, when early attacked, do not open to liberate

the seed ; saprophytic and weakly parasitic bacteria and fungi eventually cause them to blacken and rot. The white mycelium of the parasite may often be seen as a felt on the fruits, but is not usually visible on the stem.

In South India, not only are the fruits and bark affected, but the leaf fall and die back have assumed serious proportions. The leaves of *Hevea* fall naturally in December and January, but in diseased trees there is a second fall in the rains. The branches from which these leaves fall

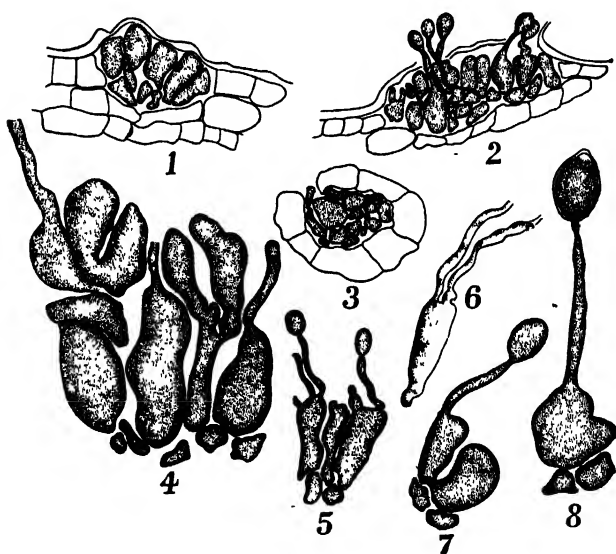


FIG. 200. *Phytophthora Meadii*: 1, stromatoid body in epidermis of fruit,  $\times 480$ ; 2, same forming sporangia,  $\times 215$ ; 3, same in tangential section,  $\times 480$ ; 4-8, cells of same, some bearing sporangia,  $\times 480$ .

often die back in the following cold weather, and fresh shoots that appear in February below the dead parts, begin to wilt in March. Tiny drops of latex may show on the leaves and leaf-stalks ; and the leaflets shrivel, dry up, and fall off. The shoot ultimately dies back to the parent branch. The fruit rot begins early in the monsoon and, soon after, leaf-shedding becomes marked. Some affected trees lose all their leaves, others only

a portion. The new flush that replaces them remains normal during the following cold weather, except where dying back is going on.

On the leaves, the first attack may be on the blade, the leaflet stalks, or the main leaf stalk. On the green leaf, dull grey spots, at first circular, then enlarging and running together, are seen. The stalks show discoloured spots, sometimes sunken. Sporangia are formed especially around the edges of the spots. The young leaf buds are liable to attack, and twig infection may start from such buds as well as through the stalk of infected fruits.

In the twigs, there is a clear line of demarcation between the dead and living parts, dark brown in colour. Hyphae occur in the dead parts and extend into those still living. They are found especially in the medullary rays and also in the other parts of the wood, the pith, and the bark.

In the stem, the mycelium of the parasite is chiefly between the cells of the soft tissues, avoiding both the sclerenchyma and the wood cells. In the fruit, intercellular and intracellular hyphae are found in the rind, and they may reach the interior of the woody shell through the sutures. The cell walls of infected tissues are turned brown or yellow and are sometimes swollen. The cell contents are destroyed and sometimes replaced by a yellow or brown, gummy substance. The hyphae are of the usual Phycomycete type but difficult to see without reagents; haustoria are rare and of a simple type, roundish or elongated (Fig. 201, 1).

When about to fructify on the fruits, the hyphae form a stromatoid body of swollen cells, not laterally united to one another, in the cells of the epidermis (Fig. 200, 1). These cells enlarge and rupture the cuticle and at the same time give out, from their free ends, from one to three slender processes, the sporangial stalks (Fig. 200, 2). The stalks may be extremely short, or up to  $100\mu$  long, and they sometimes branch so as to produce a superficial mycelium. The sporangia are usually single and in culture measure from  $25$  to  $51\mu$  in length by  $19$  to  $37\mu$  in breadth, though seldom exceeding  $35$  by  $25\mu$  when measured direct from the host plant. They are pear or citron-shaped and each gives rise to a number of zoospores (up to 30) of the usual Phytophthora type. On germination, the zoospore gives one or more germ-tubes, which may again bear small secondary sporangia with one to three zoospores (Fig. 201, 2).

Chlamydospores similar to those of *Phytophthora parasitica* are formed on the host plant and in culture (Fig. 201, 3). They are roundish,  $17$  to  $34\mu$  in diameter, terminal, lateral, or intercalary, and with a thick, usually yellowish wall.

The sexual form of fructification has not been found on the Burma fungus but has been obtained in South India in cultures, and on fruits and seeds towards the end of the fruiting season. It resembles that of *Ph. Colocasiae* in all essentials (Fig. 201, 4). The oogonia and antheridia arise on lateral branches from the main hyphae. The antheridium is hyaline and persistent, and is penetrated by the stalk of the oogonium. The latter is roundish and usually thin-walled, smooth, and colourless, but may have a thick, roughened, yellowish wall. The oospore is round, colourless or pale yellow, and nearly

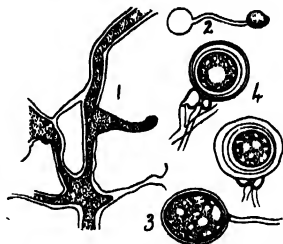


FIG. 201. *Phytophthora Meadit*: 1, haustoria on mycelium in stem,  $\times 366$ ; 2, germinating zoospore with secondary sporangium,  $\times 480$ ; 3, chlamydospore  $\times 300$ ; 4, oospore,  $\times 800$ .

kills the oogonium. Thin-walled oogonia measure from 22 to 31 $\mu$ , thick-walled 32 to 43 $\mu$ , and oospores 22 to 28 $\mu$ , average 24.5 $\mu$ .

Artificial inoculations show that the fungus is a wound parasite on Hevea stems but fails to penetrate the unwounded bark, whether of old or young trees. The first signs of attack may be visible in four days, and typical vertical cracks, with an exudation of latex, in a week. In deep-seated inoculations, gaping wounds exposing the wood were obtained. It has also been successfully inoculated on leaf blades and stalks, leaf buds, and fruits. Inoculations on cacao, papaya, and breadfruit (natural hosts of *Phytophthora Faberi*) failed, as did also those on seedlings of brinjal and tomatoes, plants which have been successfully inoculated with the cacao fungus.

The disease makes its appearance soon after the rains set in and disappears after the close of the monsoon. It seems to be closely dependent on climatic conditions, and is fostered by humid, stagnant air within the plantation.

Hence the chief treatment should be in the directions of securing good ventilation, and of protecting the newly-cut tapped surface from infection. Thinning the trees is strongly recommended or, where shade is still too dense, heavy pruning. New infections are said to come chiefly from the fruits, and the fungus does not seem to live over from one season to the next on the bark. Hence it is recommended to collect and destroy as many fruits as possible, in badly affected areas, early in the monsoon. It may be necessary in places to cease to tap infected trees during the monsoon, while where tapping is still carried on, the exposed surface may be coated with some preservative. Excellent results are said to have been obtained in South India by a combination of cessation of tapping on infected trees, with the application of a thin smear of a mixture of tar and tallow to the diseased spot. The mixture is applied with the finger and then rubbed with a piece of sacking so as to cover the attacked part of the bark. The tar acts as an antiseptic, while the tallow forms a waterproof covering. After the monsoon, the treated areas gradually shed a thin scale of tar-coated bark and expose a clean, healthy surface beneath. Covering the tapped surface with a mixture of clay, cowdung, and sulphur has also been recommended; while in Java and Malaya, antiseptics such as carbolineum and izal have been used with success.

In South India, the fungus is believed to pass the dry season inside the branches that have died back, as well as by the oospores which form



on the fruits and seed towards the end of the monsoon. It is recommended to remove and burn all dead wood and decayed fruits on the trees and on the ground. Extensive experiments to test the best methods of doing this are in progress.

**Pink disease** (*Corticium salmonicolor* B. & Br.).—So far as is at present known, this is one of the worst diseases of *Hevea* rubber in southern India. More than 2 per cent. of the trees on some estates have been attacked by it in a single year. It is also known in Burma, Ceylon, the Malay Peninsula, Java, Borneo, Formosa, Samoa, the West Indies, Surinam, West Africa, and the Caucasus. In South India, it has been found on tea, coffee, orange, mango, jak (*Artocarpus integrifolia*), camphor, Crotalaria, and *Cassia mimosoides*. In other countries, on tea, coffee, cacao, rubber (*Hevea*, Castilloa, Ficus), cinchona, indigo, rhea, nutmeg, pepper, orange, mango, custard apple, sapodella, loquat, plum, apple, coca, cinnamon, kola, *Tephrosia purpurea*, dadap (*Erythrina* sp.), *Grevillea robusta*, Strobilanthes, Lantana, Eriodendron, jak, teak, acacia, *Bixa orellana*, Thuja, Eucalyptus, jasmine, rose, gardenia, oleander, croton, and many other wild and cultivated plants. It has therefore, perhaps, a wider range of recorded hosts, belonging to the most diverse families, than any other tropical parasitic fungus yet known.

The amount of injury which this fungus is capable of causing varies greatly according to the variety of plant attacked. Tea suffers less than cinchona and cinchona less than cacao; *Hevea* seems to be one of the more susceptible plants, and *Grevillea robusta* is also very readily attacked; while Lantana frequently escapes. Experiments in Java prove that the disease can pass readily from one species of plant to another; cinchona has been successfully infected from tea, coffee, cacao, *Hevea*, loquat, and several other plants, and there is no indication in these experiments of the existence of biological races (see p. 61).

The disease affects the twigs or smaller branches chiefly, but is found also on the main stem. The first symptom noticed may be that the leaves of one or several branches lose their green colour and wither, often without being shed. In some hosts, the small branches wither rapidly when attacked, but in others (tea, etc.), death is slow. On affected branches, one or other of the several forms in which the parasite occurs can be found, but it is rare to find all stages on the same twig. The most easily recognised and most destructive is the *Corticium* stage, which occurs as a rose-coloured or whitish crust (Fig. 203, 1), often much

cracked and somewhat like badly-dried whitewash, on the under side of horizontal branches and wholly or partly surrounding those that are vertical.

On Hevea, the disease generally originates at the fork of a tree, or where several branches arise close together from the main stem. The Corticium stage of the fungus is usually the most readily observed, forming a pink incrustation on the bark. This gradually extends and may ultimately surround the stem and the branches arising from the affected part for a length of several feet. The bark then splits and peels away from the wood, and the latter may also become infected, but to a much



FIG. 202. Pink disease of rubber (*Corticium salmonicolor*): 1, Hevea twig with incrustation,  $\times \frac{1}{2}$ ; 2, section of sporophore showing hymenium,  $\times 220$ ; 3, basidiospores germinating,  $\times 195$ ; 4, Necator stage,  $\times 200$ ; 5, Necator spores,  $\times 200$ ; 6, same germinating,  $\times 200$ . (2-3 after Zimmermann, 4-6 after Brooks and Sharpley).

less extent and chiefly in the smaller branches. Even after the bark has been killed, the fungus spreads more rapidly over the surface than within the tissues, so that the margin remains generally superficial. Spread is governed largely by conditions of moisture or exposure; if, for example, the rain water runs down one side of the stem only, the crust may be

confined to that side ; and similarly it will exhibit greater growth on the more shaded side. The pink layer is extremely thin, and, when old, splits everywhere in lines more or less at right angles to one another. For this reason it has been called the "writing fungus" in Malaya, being thought to resemble hieroglyphics. Old specimens lose their pink colour and become ochraceous, or, when very old, bleached to white. Sometimes even young patches may be white, and the bark may be covered with long, silky hyphæ of a silvery white appearance instead of the usual interwoven pink felt. This stage is sufficiently common on other hosts, such as cinchona, to merit being considered a definite sterile form of the fungus, somewhat like the sterile sheets of *Corticium* found on coffee twigs. Another sterile form also occurs, often in connection with the last, as small, white nodules partly sunk in the bark, and often elongated or in rows. Finally, there is a spore-bearing form, which was long considered to be a distinct fungus under the name of *Necator decretus*, but which has been proved to belong to the other forms. It appears as irregularly rounded or elongated bodies, sometimes superficial, sometimes bursting through the bark from below, orange-red in colour, from  $\frac{1}{2}$  to 2 or 3 mm. in diameter, and often closely crowded together.

The amount of damage caused depends upon the size of the tree. The disease appears to attack *Hevea* stems mostly of between one and three years old, and after that to be chiefly confined to the upper branches. Young stems about two years old are quickly encircled and "ringed," but on older trees, the patch should be detected before it has grown completely round the tree. When the penetrating mycelium reaches the laticiferous tissues, an exudation of latex frequently occurs, which assists in indicating the presence of the disease even high up on the tree. Growth takes place continuously during the monsoon period ; the bark is killed off uniformly ; side branches at the point of attack are ringed and killed ; and the bark of the main stem peels off in large patches. Before the dry weather comes, the growth of a single season may be enough to encircle the stems of young trees, and the whole stem above may then be killed. But in many cases the disease has not advanced far enough to kill the tree by the time the rains cease. The fungus then stops growing, having killed off part of the cortex and cambium of the main stem and probably some of the side branches also. This leaves an open wound, over which there is no cambium to produce new tissue. The result is a "canker"—an open wound exposing the wood and surrounded by an elevated callus ring. The pink incrustation does not form on the

exposed wood, and sometimes that on the bark has been for the most part thrown off with the dead bark when the canker is formed, but it is usually possible, even in this stage, to find traces of the pink fungus on the fragments of dead bark round the margins of the wound. Large open wounds on the upper part of *Hevea* stems are generally the result of the attacks of this fungus. Adventitious roots have been observed to develop from the callus of the upper side of these wounds on hosts where the stem was partly "ringed." Below the point of attack, the tree usually throws out a bunch of suckers in its endeavour to replace the part killed, and these often serve to call attention to the disease.

The mycelium of the parasite consists in the early stages of fine, white hyphae which creep over the surface of the bark, sometimes condensing to silky, white sheets, sometimes remaining thread-like. Clamp-connections occur in these hyphae. The next stage to be observed is usually the appearance of the white, nodular form, which, on close examination, is found to occur chiefly in the lenticels. It is suggested that entrance into the tissues occurs in this stage. The nodules are composed of dense coils of fine colourless hyphae, which communicate both with those on the surface of the bark and those which enter the tissues. In their neighbourhood, the cells are killed and turn brown, and this appears to represent the first stage of the attack. When the wood is invaded, the hyaline hyphae are found especially in the vessels and medullary rays. Thylogae are found in numbers in the affected vessels (Fig. 44). The ascent of water is checked, and the leaves above wither and die.

The Corticium stage is formed of a loose, basal layer of interwoven hyphae, which unite into a much firmer basidial layer, composed of a dense mass of parallel basidia, at the surface (Fig. 202, 2). The individual basidia are club-shaped, with 4 spores borne on narrow sterigmata, 4 to 6 $\mu$  long. The spores are hyaline, pear-shaped, with a little stalk at the narrow end, and measure 9 to 12 by 6 to 7 $\mu$  in diameter. They germinate readily in water (Fig. 202, 3). In Malaya, such a definite hymenial layer as that figured has not been found, the basidia being irregularly scattered. The Corticium stage is usually preceded by the white, nodular stage, but seems to have no relationship in time with the Necator stage and often occurs without the latter.

The Necator stage is usually found only on twigs on which the Corticium stage also occurs, but is sometimes found alone. In artificial inoculations with pure cultures from the Corticium, only the Necator or the Corticium may appear, or both together: the same occurs when the cultures originate from the Necator stage, and there seems to be no rule which would enable one to predict what form the parasite will take. The fruit-bodies would seem usually to arise from the internal mycelium and burst out through the bark in little groups. On horizontal branches they occur mostly on the upper surface, unlike the Corticium which is usually underneath. Each fruit-body consists of a roundish, stromatic mass of pseudoparenchymatic cells of an irregularly polygonal form (Fig. 202, 4). The diameter is  $\frac{1}{2}$  to 2 or 3 mm. and the colour of the whole body orange-red. After bursting out to the surface, the outer part of the cell mass breaks up into unicellular spores (Fig. 202, 5). This process continues into the deeper layers of the stroma, until most of its contents have been transformed into spores. The spores are hyaline singly, reddish in mass, irregularly angular in shape, very variable in size, from 10 to 35 by 7 to 17 $\mu$  in diameter, and of a waxy consistency. They germinate readily in water, forming one or more germ-tubes (Fig. 202, 6), which give rise to a richly-branched, septate, anastomosing mycelium.

Artificial cultures of all four forms, the sterile creeping mycelium and nodular forms and the fertile Corticium and Necator stages, quite agree in their characters. They all

give a copious mycelial growth, which is usually coloured pale rose if exposed to the light, but remains white, at least for a time, in darkness; other conditions, such as the composition of the culture medium, and the position of the hyphæ on its surface or immersed in it, also influence the colour, but light seems to be the chief and its effects may be seen in aporphores of the Corticium stage, which are often white or pale in shaded positions. The colour is due to the presence of carotin. If the cultures be exposed to rapid alterations of temperature, zones develop. No form of fructification has been obtained in culture, the fungus giving only a mycelial growth with sometimes sterile nodular condensations of the hyphæ, which may resemble the Necator stage before spore production.

Infections carried out in Java with pure cultures, taken from the different forms of the fungus, show that all its forms behave in a similar fashion, successful results being as readily produced by the culture from one stage as from another. It is not a wound parasite, being capable of infecting through the sound bark. Moisture is, however, essential; it is exceedingly difficult to get successful infection in dry weather, and it is, further, probable that the spores are only formed in the moist season of the year. The commonest seat of infection is the fork of a branch or stem, this being the dampest part of the bark. Even after infection has occurred, a dry period will check the further development of the fungus towards its fructifying stage. The moisture conditions depend not only on the season, but are also influenced by the density of planting and the use of inter-crops, cover plants and the like. It has been conclusively established that the prevalence of not only pink disease but also canker is very largely bound up with the extent of the shade, the density of the growth, and, in general, the degree of moisture and stagnation of the air within the plantation. This is very clearly shown in the case of certain species of *Cinchona* with different habits of growth. *C. Ledgeriana* has a rather dense, bushy growth, and suffers severely from pink disease; *C. succirubra*, on the other hand, has twigs which stand rather far apart and allow the air to circulate more freely, and in consequence is rarely injured. Artificial infections have shown that this is not due to any difference in the power of the two species to resist the disease. For the same reason, tea in seed nurseries is more liable to attack than the pruned tea of the general crop. Shade is said also to have possibly a more direct influence on the prevalence of the disease than merely by producing a humid atmosphere; it is suggested that changes in the composition of the cell sap brought about by shade (increase of plant acids especially) assist in facilitating the penetration of the hyphæ.

Hence, of the measures for reducing the losses caused by this fungus, mention should be made in the first place of the beneficial effects of

promoting a free circulation of the air by reduction of shade, thin planting, avoidance of inter-crops, and the like. Good drainage is also of value in the same direction. These are matters fully dealt with in many of the publications on rubber cultivation in general, and need only be emphasised in the present connection.

The direct treatment may be divided into measures intended to save already infected trees, and measures to prevent infection.

A close watch should be kept at all times, and especially from May onwards, for the appearance of the disease on the trees, and when it is found, the affected part should be cut off at least eighteen inches below the attack, and burned. Attempts made to cut out the affected bark only, even when the wound has been washed with Bordeaux mixture and tarred, have generally failed, about 70 per cent. of such treated areas having developed the disease again the following year.

Consequently the whole branch or stem should be cut off. Unless three feet of tappable stem can be left, it is better, in an attack on the main stem, to cut the tree down close to the ground and get a sucker from low down to replace it. The diseased parts should be burned on the spot at once, to prevent dissemination of spores, and the knives and chisels should be washed in a disinfectant (permanganate of potash is generally used) after dealing with each case; the coolies should also wash their hands in the disinfectant before going on to the next tree. All the wounds and cut surfaces of branches should be tarred, not necessarily as a protection against this fungus but to prevent infection by wound parasites, such as that which causes the die back disease (see p. 509), and also, when the heart-wood is exposed as when the stem or large branches are cut, to prevent the access of various common wood-rotting organisms.

To prevent new infections, the use of Bordeaux mixture has been found very successful in young rubber. It is certain that the beginning of the attack is due to spores, whether from jungle trees or from the rubber itself. It is believed that the spores do not survive the dry season, and that new ones are not formed during this period. on rubber, so that the attack regularly noticed on certain estates very quickly after the monsoon begins, probably comes from some external source. Hence if the parts of the tree most susceptible to attack, such as the forks of the branches, can be coated with Bordeaux mixture before the onset of the monsoon, the spores lodging in these places will be killed. If the mixture

can be made to stick on the tree during the monsoon, one application should result in keeping off the disease for a whole year.

The strength of Bordeaux mixture used in successful experiments on a large scale, in 1910-11, was 6 lb. copper sulphate, 4 lb. lime, and 45 gallons water. To increase the adhesive powers of the mixture, the resin adhesive (see p. 142), is now always used in rainy places in South India, but in the experiments referred to, treacle and other inferior substitutes were tried. The mixture was kept well stirred in wooden or copper vessels and given to the coolies in small lots in hollow bamboos. The coolies climbed the trees (a six-foot ladder was used in some cases) and applied the mixture with a brush round the forks and for a foot or two down the stem and up the branches. In some cases, the stem was rubbed clean with old sacking first. The best time for applying the mixture is said to be May or June, and a second application is probably unnecessary, but subsequent experience may show that further treatment during a break of the monsoon in September or October may be advantageous. The cost was from one to two rupees an acre, on over 200,000 trees treated in 1910-11, and the result was a reduction of the disease by from 50 to 75 per cent. Some of the trees had three applications, some two, some only one. The results were about the same in the two latter cases, but those done three times had less disease. Further experiments are, however, necessary before deciding on the number of applications which gives the best result.

It is evident that, if further experience supports these very encouraging results, a cheap and effective method of controlling this disease in young plantations is available. In young rubber, cutting out the affected parts, though undoubtedly effective in saving many trees, may mean the loss of three or four years' growth, which is a very serious matter in a crop that takes so long to give a return. It is not likely that the use of Bordeaux mixture will ever entirely do away with the necessity for surgical treatment in a certain percentage of cases, but with this crop prevention is so obviously better than cure, that it is to be hoped that the preventive treatment will, if it stands fuller tests, be adopted where pink disease is prevalent. It must be clearly understood, however, that the method cannot cure a tree already diseased, and that many consider Bordeaux mixture to be unsafe after tapping has begun. The part of the fungus within the tissues escapes injury, and at a subsequent period can renew the spread of the disease on the surface as well as below. In Malaya, it is recommended to apply tar for two feet above and below the

region over which the fungus is apparent, in early stages of attack. The treated trees should be examined again within a month, and a further application made if the fungus has spread. If this fail, the branch should be cut out. Spraying is not recommended in mature plantations, owing to the difficulty of reaching the upper branches.

The above account refers to pink disease chiefly as it occurs on *Hevea*. It attacks also *Castilleja* and *Ficus elastica*, though it has not yet been noticed on these hosts in India. In both cases the damage seems to be similar to that caused to *Hevea*. Another interesting host is *Crotalaria striata*, grown for green manure and cover in many estates. In South India, large plants of this species, about one year old and  $\frac{3}{4}$  inch thick, and very woody at the base, were freely attacked at the lower part of the stem by pink disease. But this crop can be safely grown as a rule, provided it is not allowed to develop such woody stems. The same applies to *Cassia mimosoides*, another cover crop.

**Brown root disease** (*Hymenochaete noxia* Berk.).—This is the only rubber disease of the class which take their origin from rotting stumps of jungle trees in the soil, so far identified in India. In Ceylon and Malaya, various other root diseases of this type are known to attack rubber. In particular, a disease caused by a fungus usually known as *Fomes semitostus* Berk. is regarded as one of the worst enemies of rubber cultivation in the Malay Peninsula; but though the fungus first described by this name occurs in the Khasia Hills, it is now clear that the rubber parasite is not the same; the latter, which should be known as *Fomes lignosus* Kl., has only once been reported in southern India, and it has been included amongst the pests against the importation of which legislative measures have been adopted. In Ceylon, *Hymenochaete noxia* is the commonest root parasite of rubber. Other root diseases are caused by *Sphaerostilbe repens* and *Ustilina zonata*, but these have not been identified in India on this host, though known on tea.

The characters of the disease, as it affects tea, have been given above on p. 429. On rubber it is quite similar. There is reason to fear, however, that the damage which this and allied diseases may ultimately cause in rubber estates is likely to be more serious than in any other class of cultivation. Mycologists in the East, especially in Malaya and Ceylon, have frequently called attention to this danger, which results largely from the peculiar conditions under which rubber plantations have been often opened in heavy jungle. At the time of the rubber boom, large areas were hastily cleared in a very imperfect fashion,



especially as regards the larger jungle trees. These were for the most part not uprooted, but cut off, usually 2 or 3 feet above the soil level, and left to decay. Even the fallen timber was often not removed (Fig. 204). In such estates, root diseases began to appear rapidly, sometimes when the rubber was only two or three years old. In one instance reported from Malaya, 10 per cent. of the *Hevea* had been killed out by *Fomes semitostus* before the estate was four years old. Another case is reported from Ceylon where 700 trees blew over from the same cause, in a single night, in an area of 80 acres, after which all dead stumps and fallen timber were taken out. It is possible that new outbreaks of root disease will continue to appear in such estates for many years. It has already been stated that, in coffee, there is reason to believe that certain classes of stump rot do not begin to develop until as much as twenty years after the jungle tree, from which they originate, was cut down. The history of rubber estates in the East is as yet too short for a similar experience to have been recorded; but the possibility, even probability, must be faced that, in imperfectly cleared estates,



FIG. 203. Brown root disease of rubber (*Hymenochaete novia*): affected root of *Hevea*,  $\times 4$ .

new outbreaks of root disease will continue to develop for many years.

The clearing of old stumps and fallen timber from newly opened-up rubber estates was not done at first, partly on the score of expense, partly because the danger was not recognised. But experience has shown that the ultimate cost of such negligence has often greatly exceeded what would have been the price of prevention in the first instance, and in some cases the real loss has been irreparable. It is far more difficult to clear estates after they have been planted out, and it is impossible to guarantee



FIG. 204. View in Malayan rubber estate, showing stumps and fallen timber.  
(After Leprieu).

that the cleaning up will be effective in checking disease once it has started. From the mycological point of view, the path of safety lies in thoroughly clearing and cleaning an estate before, and not after, planting operations. From the business aspect, it seems probable that this means economy certainly within ten or twenty years. At the annual meeting of one Company, in 1910, it was stated that the expense of uprooting

stumps and removing all dead wood came to a total charge, once for all, of less than sixpence for each tree. This is not a heavy outlay to meet a danger which has become so marked that stringent legal measures against negligence in cleaning rubber estates have actually been advocated in the Straits Settlements.

The treatment of brown root disease, once it appears, has already been discussed under "tea."

**Die back** (*Thyridaria tarda* Bancr. = *Botrydiplodia Theobromæ* Pat.).—A description of this parasite has already been given with an account of the diseases of sugarcane and tea caused by it (pp. 385, 445). On rubber, it is as a parasite of the twigs and branches that it is best known, and the name "die back" is descriptive of the predominant symptom, namely the death of infected parts backwards from the tip towards the base. It has been most frequently observed on *Hevea*, but serious outbreaks on *Castilloa* have also occurred. The fungus itself has been found on surface roots and cut stems of *Ficus elastica*, but its pathologic action has not been investigated on this species. As a rubber disease, it has been chiefly studied on *Hevea*.

In young *Hevea* plants, the main shoot is usually attacked; in older plants, the leader or one of the lateral shoots. At first, it is rare to find more than one or two shoots dying. The death of several shoots occurring simultaneously is usually due to some other cause, such as root disease.

When a branch is attacked, infection usually takes place not at the tip, but a variable distance back from the growing point: the branch dies above the point of attack and, less rapidly, also backwards towards the base; ultimately the trunk and even the roots may be reached. As spread backwards occurs, the whorls of branches lower down are killed off in succession, owing to stoppage of the water supply where they arise from the stem. New shoots are often put out below the dead part, and these in turn may also be killed. It not unfrequently happens, however, that the dying back of the branch or stem is checked, and that growth is continued by one of the shoots which has arisen lower down. This arrest of the disease is, at times, noticeable in vigorous trees at the point where a diseased branch joins the main stem.

In some cases, the disease appears to originate in the roots. This has been observed in recently planted out "stumps," in Ceylon, Malaya, and Burma, in one case in Burma causing the death of 60 per cent. of the stumps. The fungus, which is almost always present in the soil, appears

to gain an entrance through injuries caused during the period of exposure of the roots prior to planting out, though in some cases "basket" plants (that is, seedlings planted out in the small baskets in which they have been raised, and therefore not exposed to injury at the roots during planting) have been attacked. It is also suggested that older plants may become infected through exposed lateral roots above ground, but this appears to be rare.

The cases of disease in an estate are usually scattered, not in groups. They are most numerous and the damage greatest, as a rule, in areas where the plants are in some way unhealthy. In old rubber, where the trees have scanty foliage and are of slow growth, whether from insufficient sunlight, excessive tapping, defective soil conditions, or other causes, the disease is said to be most common. In Ceylon, however, where die back is said to be due to the combined effect of this parasite and another known as *Glaeosporium alborubrum*, young trees, one to two years old, are said to be most frequently attacked. "Stumps" are also very liable to attack, both through the cut end of the stem and through the roots.

The progress of the disease is rapid. In Ceylon it is said to kill the tree in a month or six weeks after death of the uppermost branches is noticed. In the Federated Malay States, artificial inoculations caused the death of five months' seedlings in about three weeks, while one-year-old plants took eight or nine weeks to die; in both cases inoculation was not necessarily fatal, some plants succeeding in checking the disease after it had spread backwards for a certain distance.

As previously mentioned, the fungus is primarily a parasite of the wood. The medullary rays turn brown early in the disease; later, the whole wood assumes a greyish colour, ultimately darkening considerably. The outer tissues are attacked later than the wood, and latex may continue to be given for some time after the underlying wood has become diseased. Ultimately, however, the cortex is invaded, the bark turns grey, cracks, and peels off, and the cambial layer between the bark and the wood is reduced to a black mass, produced by the growth of the dark coloured mycelium of the fungus. There is usually a sharp line of demarcation between dead and recently infected tissue in the wood, as the disease is traced downwards along the branch.

The mycelium of the parasite is wholly internal. Large, brown hyphae are found in the tracheids, running longitudinally and sparingly branched. Others give a more abundantly branched growth in the medullary rays. They pass from cell to cell through the pits in the walls, when these are present, being much constricted at the point of passage; except at this point, the cell walls are not dissolved. Starch is destroyed in the affected storage cells. The cambium cells have their contents exhausted and the tissue is converted into a black mass, composed chiefly of hyphae. The phloem and cortex are similarly invaded at a later stage. The hyphae in the wood are always in

advance of those in the outer tissues, and may be found four or five inches below the point where the wood appears dead to the naked eye.

Pycnidia begin to appear on the dying branches after the death of the outer tissues; they have been found after eleven days in some artificial inoculations. On the younger parts, they are scattered, showing as very minute, black points sunk in the bark. On older parts, they tend to be aggregated together in stromata. They have already been described (p.387). An Ascomycete, *Thyridaria tarda*, is believed to be the perfect stage of this fungus, but only develops on old stromata some months after the death of the branch in which they occur. The proof of its genetic connection with the Botryodiplodia is not conclusive.

Inoculation experiments on *Hevea* prove that the spores from the pycnidia can infect the plant through wounds only, producing symptoms exactly similar to those observed in natural outbreaks. Unwounded surfaces are not attacked. The wound must be deep enough to expose the wood. Properly tapped surfaces, where the cambium and secondary phloem bordering it are not killed, do not admit of infection. Any dead surface involving the wood, such as a dead branch, will allow the parasite to enter. The following are some of the chief channels of infection. In the planting out of stumps, the cut ends of the stump may be infected and also infection may occur below ground on injured roots: when growth commences again, a shoot arises below the cut end, and the latter dies down to the point of origin of this shoot; the dead end can admit the fungus. Topping, pruning, wind, animals, and the like, cause wounds exposing the wood; the lower branches also die as the tree grows older; infection readily occurs in such cases. The highest branches sometimes die, especially in badly drained or poor soils; such dead branches permit the fungus to enter. Pink disease and black thread may also kill the outer tissues, and are frequently followed by die back.

The parasite extends in dead or dying tissues far more rapidly than in living. After a branch has been infected some distance behind the apex, and begins to die above the point of infection owing to stoppage of the water-supply, the hyphæ run upwards into the dying part much faster than downwards into the still living tissues. Unhealthy tissues are, therefore, more liable to injury by this fungus than healthy, and, in general, it is said that the further the plant is from its full condition of vital activity, the more liable it is to the "die back" disease. Hence it has been found frequently to follow on attacks of root disease caused by *Fomes lignosus* or *Hymenochaete noxia*. In Ceylon, it is said usually to follow an infection of the green shoots by *Glæosporium alborubrum*.

- The methods of treatment are based on the above considerations.

As the fungus grows and fructifies freely on dead and dying wood, not only of rubber, but of a number of other plants, proper sanitation of an estate necessitates the collection and burning of all fallen wood and branches, which should not be left lying about to rot.

A healthy growth of the plants should be secured by good cultivation, avoidance of close planting and excessive tapping, good subsoil drainage, and the like, since the parasite is less able to damage vigorous trees.

Diseased branches should be removed by cutting back until sound wood is reached, which is about six inches below the lowest point at which discoloration of the wood can be noticed. A sloping surface should always be left after removal. Large branches should be taken off flush with the stem. It is recommended for this to make three cuts: one, eight or ten inches from the trunk, from below upwards half through the branch; a second, a couple of inches further out, from above downwards until the branch breaks off or is sawn through; and finally, a third, to remove the stub flush with the trunk. A smooth surface should be left. Branches that die from any cause should be similarly removed.

All wounds which expose the wood should be tarred, whether they are from pruning, removing dead branches, excising diseased parts, or accidental. The cut ends of "stumps" should especially be protected. Coal tar is recommended in preference to Stockholm tar, as being more permanent and offering a better protection against fungi. The tar should be applied in a thin layer with a brush. The cut surface should be allowed to dry slightly before it is tarred, and the tar should not be allowed to run down the side of the branch or trunk.

"Topping," and deep tapping with consequent exposure of the wood, should be avoided. So also should any danger of killing the bark, as by scorching from burning prunings too near the trees.

As the fungus is universally present in estates, both in the air and in the soil, reliance must be placed chiefly on promoting a vigorous growth of the trees and on protecting cut surfaces, which expose the wood, against its attacks. That it has not caused more damage is, perhaps, due to its inherent lack of virulence as a parasite, but it seems to vary in this respect and may in the future be found, especially if provided with an abundant supply of living food, as in large estates, to develop an accentuation of virulence sufficient to make it a serious pest.

**Anthracnose** (*Glomerella cingulata* (Stonem.) S. and v. S. - *Neozimmermannia Elastica* Koord.=*Glaeosporium Elastica* Cke. & Mass.=

*Colletotrichum Ficus* Koord.).—This parasite, which is amongst the commonest fungi found on the leaves of sub-tropical evergreen plants, has already been referred to as causing diseases of tea, and probably chillies and coffee, in India. In the United States, it has been found on over 30 different hosts and has been shown to be of great economic importance. It occurs, in rubber-yielding species, chiefly on *Ficus elastica*, but has also been found on *Hevea*. In India, we only know of it as yet from Burma. Ceylon, Java, East Africa, and the Philippines are the other countries from which it has been reported as a rubber disease; while in Europe it has been found on *Ficus elastica* in glass-houses, no doubt introduced from abroad.

The fungus has long been known on *Ficus* in its conidial stages, of

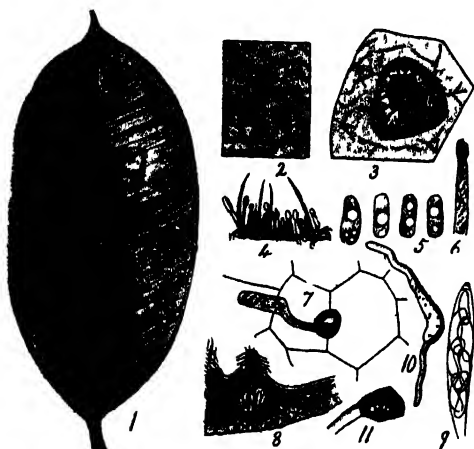


FIG. 206. Anthracnose of rubber (*Glomerella singulata*) 1, affected leaf of *Ficus elastica*,  $\times \frac{1}{2}$ ; 2, part of same enlarged to show acervuli; 3, an acervulus, more highly magnified; 4, same in section; 5, conidia; 6, young conidium on stalk; 7, germinated conidium with appressorium; 8, perithecialium; 9, ascus; 10, germinating ascospore; 11, tip of germ-tube from an ascospore, with appressorium. (8-12 after Koorders).

which there are two, formerly known as *Glaoesporium Elasticæ* and *Colletotrichum Ficus*. As already mentioned, these two genera of the Melanconiaceæ are not sharply separated from one another, most species of *Colletotrichum* occasionally appearing as *Glaoesporium*, and *vice versa*. Hence it is, perhaps, better to consider the fungus as having a single

conidial stage, which sometimes has sterile setæ and sometimes has none, this being the only distinctive feature of the two forms.

The conidial stage causes the appearance of large, irregular, discoloured patches on the leaves, the biggest sometimes several inches in length; these are often marginally placed. At first the spot is brown; later, the central part is grey, the margin darker, and there is usually a light yellow band where it joins the healthy tissue. There may be several smaller spots on a leaf, or a single large one, usually situated to one side. In the older part of the spot, the conidial beds may be seen as minute dots formed on both surfaces, sometimes in distinct concentric circles (Fig. 205, 2). The dots are usually blackish, but when spore production is copious (as in moist weather) may have a pink tint.

The mycelium occupies all parts of the withered tissues. The hyphæ collect in stromatic masses just under the epidermis and here the spore-beds are formed. By the pressure of the continued formation of spores, the epidermis is ruptured and the spore-bed exposed (Fig. 205, 3).

The spore-bed is formed of dark brown, septate hyphæ, united into a disk-shaped stromatic layer of pseudoparenchymatous cells. From this, rows of short conidiophores ("basidia") arise in a crowded layer, each being unbranched, unseptate, colourless (or somewhat brown at the base), rounded above, and 7 to 8 by  $3\mu$  in diameter (Fig. 205, 4). Sometimes, at the margin, a greater or less number of dark brown, rigid, sterile bristles ("setæ") are found. According to their presence or absence, the fungus may be considered a *Colletotrichum* or a *Gloeosporium*. They are very thick-walled, broader below, narrowed to a point at the tip, which is lighter in colour than the rest, 0 to 3-septate, and 30 to 90 or rarely up to  $160\mu$  long, by  $3\frac{1}{2}\mu$  broad.

The conidia (Fig. 205, 5-6), are borne singly at the tips of the basidia. They are non-septate, colourless (but pale rose in mass), cylindrical or sometimes a little narrowed at the ends, straight or very slightly curved, and mostly 10 to 15 by  $3\frac{1}{2}$  to  $4\mu$  in diameter, though larger and smaller spores can be found.

They germinate readily in water, often dividing first into two cells by a median septum, each of which usually gives out a germ-tube. The further growth from the germ-tube varies according to circumstances. When sown in water or on a leaf of the host plant, the hypha branches little and forms at its tip, after a longer or shorter growth, a thick-walled, deep brown or black cell, exactly similar to the chlamydospore-like bodies already described in *Colletotrichum falcatum* from sugarcane (Fig. 205, 7). As in the latter case, they have a double function, being both organs of adhesion (appressoria) and resting spores. As infection of the leaf usually occurs by means of a hypha from these bodies and not direct from the conidium, it is probable that they also serve to accumulate enzymic energy to assist penetration. In well-nourished cultures, on the other hand, the germ-tube from the conidium branches into a mycelium, on which new conidia are produced, usually at the ends of lateral branches. The conidium is detached when mature and a second arises in its place; up to 15 have been observed thus successively formed. Chlamydospores may also be formed in such cultures at a later stage.

The perfect (perithecial) stage of the fungus is usually produced on dead parts of the host plant, mostly fallen leaves and twigs. The perithecia arise singly or in little adherent groups, and are small (about  $100\mu$  in diameter), round or broad ovoid, black, and sunken in the host tissues except for the rather long neck, the mouth of which opens to the surface (Fig. 205, 8). The neck, which is often from once to twice the diameter of the



perithecium in length, is provided with hairs, but the rest is smooth. The asci are cylindrical or club-shaped, measuring 42 to 65 by 7 to 8 $\mu$ , and with 8 spores arranged in two rows (Fig. 205, 9). Spores cylindrical, curved, unicellular, hyaline or very slightly tinged brown, 14 to 19 by 3 $\frac{1}{2}$  to 5 $\mu$ . There are no paraphyses.

The ascospores germinate in much the same manner as the conidia, usually dividing first by a median septum and giving a germ-tube from each cell (Fig. 205, 10). This soon ends in a chlamydospore quite similar to those described above (Fig. 205, 11), if the spores are sown in water or on a leaf of the host, but give a branched mycelium with copious production of conidia, if sown in a nutrient solution. This mycelium cannot be distinguished from that produced by germination of the conidia.

Both ascospores and conidia are capable of reproducing the disease, but the former appear to be the more virulent, artificial cultures from them proving capable of attacking quite sound leaves of young plants of *Ficus elastica*, whereas those from conidia usually require the leaf to be wounded, or the plant weakened in some way, before positive results are obtained. Infection occurs by a hypha from one of the appressoria entering the epidermis directly into an epidermal cell, whence the mycelium spreads from cell to cell through the leaf.

Besides the leaf form of the disease, young stems of *Ficus elastica* have also been observed to be attacked, and have been artificially inoculated from conidia developed on the leaf. So far as is known, this form of attack is not a serious one, though if the parasite were to prove capable of establishing itself as a stem parasite (like many of its allies), it would be capable of doing far more damage than as a leaf parasite only.

Remedial measures have not been tried. The fungus is chiefly an enemy of *Ficus elastica*, a rubber tree in which comparatively little interest has been taken of late. The disease also has not been reported as doing serious damage; but it belongs to a group which includes so many dangerous parasites, that they are never safe to neglect.

Several other allied forms have been described on leaves of *Hevea* and *Ficus*, none of which have proved important. A *Gloeosporium* has also been found parasitic on green stems of *Hevea* in Ceylon and Malaya, and is evidently very similar to the fungus described above.

**Hevea leaf spot** (*Helminthosporium Heveae* Petch).—This leaf disease is commonly found in young *Hevea* in nurseries, when the plants are about 3 feet high, in Ceylon. It occurs also in South India, Malaya, and Java.

Attacked plants show on the leaves, small, round, purple spots, which gradually turn white and are then surrounded by a brownish-purple border. They may occur in great numbers, but usually remain small, not exceeding 5 mm. in diameter.

Spores are produced on short hyphae which emerge from the leaf tissues on both surfaces of the spot. The conidiophores are scattered, unbranched, greenish-brown

and 80 to 200 $\mu$  long. The conidia are brown, boat-shaped, with 8 to 11 septa, and measure 100 to 120 by 15 to 18 $\mu$ .

No treatment seems to be required but, if it became severe, spraying with Bordeaux mixture would doubtless check it. It has not been found on full-grown trees and does not cause defoliation even of the young plants.

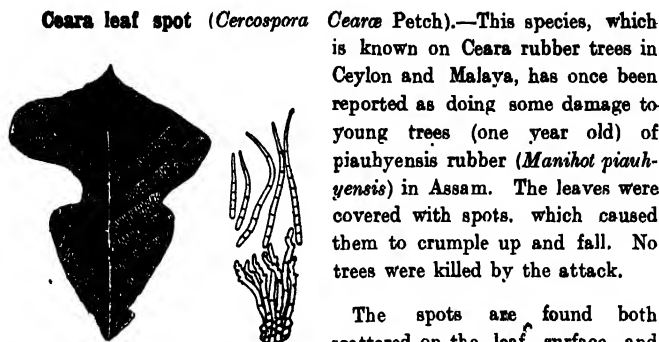


FIG. 206. Ceara leaf spot (*Cercospora Cearæ*): leaf of piauhensis rubber with spots,  $\times \frac{1}{2}$ ; conidiophores and conidia,  $\times 200$ .

*Cearæ* Petch).—This species, which is known on Ceara rubber trees in Ceylon and Malaya, has once been reported as doing some damage to young trees (one year old) of piauhensis rubber (*Manihot piauhensis*) in Assam. The leaves were covered with spots, which caused them to crumple up and fall. No trees were killed by the attack.

The spots are found both scattered on the leaf surface and united into more or less continuous bands along the margin. Individual spots do not usually exceed

$\frac{1}{2}$  inch in diameter. They are brown at first, then greyish-white in the centre, with a deep brown border.

The conidiophores emerge in clusters on both leaf surfaces, and are brown, unbranched, with numerous knee-bends towards the top, septate, and of variable length on different spots, from 40 to 100 $\mu$ , by 4 to 5 $\mu$  broad.

The conidia are almost colourless or pale olive-green, straight or curved, tapering a little towards the tip, 5 to 10-septate, and measure 40 to 110 by 4 to 6 $\mu$ .

No remedial measures have been tried, but it would probably be advantageous to remove affected leaves in the early stages of attack.

## ACKNOWLEDGEMENTS.

My acknowledgements are due to Mr. R. D. Anstead, Deputy Director of Agriculture, Planting Districts, Madras, and Mr. A. C. Tunstall, Mycologist, Indian Tea Association, for reading the sections dealing with diseases of Plantation Crops and for many useful suggestions.

To the staff of the Mycological Section, Pusa, I am grateful for much assistance in various directions.

The illustrations are mostly the work of the staff of Indian artists at Pusa, especially Messrs. R. Rao, K. D. Das, and N. Bagchi. The microscopic details are usually from copies in ink of my pencil drawings. Figures that have been previously used in publications from Pusa are not separately acknowledged, but I am indebted to my colleagues Messrs. Hutchinson, Bainbrigge Fletcher, McRae, and Shaw, and to my assistant Mr. Dastur, for permission to use several drawings and blocks. Where use has been made of illustrations from other sources, the author's name is given under the figure. I have to acknowledge permission to copy figures received from the following:—R. Maire (Fig. 4, 1): Messrs. Ch. Amat, 11, rue de Mézières, Paris (Figs. 15, 1; 42) from Ducomet's "Pathologie Végétale": U. S. A. Dept. of Agric. (Figs. 15, 5; 38, rt. fig.; 40; 118, leaf; 151; 152, 3; 183, 3): Ed., "Annals of Botany" (Fig. 18, 3-4): E. S. Salmon (Fig. 23): R. S. Troup (Fig. 30) from Ind. For., XXXVIII, Pl. III: S. Kusano (Fig. 37, 2): V. Ducomet (Figs. 37, 4; 41, 2-4; 46, lr. fig.): Ed., "Annals of Applied Biology" (Figs. 41, 1; 44; 202, 4-6) from Ann. Appl. Biol., I, Pl. XXI, and II, pp. 66, 69, 70: B. M. Duggar (Figs. 51, 2; 121, 2): Jakob Eriksson (Figs. 51, 3-4; 61, 5-6): Dept. of Agric. Victoria (Figs. 54, 3-5; 56, 3-4): Sec., Torrey Botanical Club (Figs. 57, 2 & 4; 101, 1-2 & 4; 118, conidia; 133, 2-9): Messrs. Baillière & Fils, Paris (Figs. 57, 3; 59, 1-3 & 5-7; 129, conidiophores & conidia; 173, 5-7): Kölpin Ravn (Fig. 64, 3-5): C. W. Edgerton (Fig. 103, 3): Ed., "Mycologia" (Fig. 152, 4-5): Dir., École Nationale d'Agric., Montpellier (Fig. 112, conidia): L. C. Coleman (Figs. 113; 114; 198, 8): Soc., Society for the Promotion of Christian Knowledge (Fig. 116, 1 & 3) from Ward's "Diseases of Plants": Royal Dublin Soc. (Fig. 116, 4): Acad. Roy. d'Agric., Sweden (Fig. 117, conidiophores & conidia): Dir., Connecticut Agric. Expt. Stat. (Figs. 127, 3; 141, 4): Sec., Bombay Nat. Hist. Soc. (Fig. 136, 2-4) from Jn. Bombay Nat. Hist. Soc., XXI, Pl. A, B: Dir., Missouri Bot. Gard. (Fig. 157, lr. fig.): Dir., Proefstatie v. Java-Suiker-industrie (Fig. 170): Messrs. A. Challamel, rue Jacob, Paris (Figs. 173, 5-7; 191, 3) from Delacroix and Maublanc "Maladies des Plantes des Pays Chauds": Linnean Soc., London (Fig. 173, 8-12): Ed., "New Phytologist" (Fig. 180, 2): Dept. Landbouw, Nijverheid, en Handel, Java (Figs. 181, 3; 191, 2; 196, micr. det.; 198, 1; 202, 2): Controller, H. M. Stationery Office (Fig. 183, 2) from "Kew Bulletin," 1899, No. 151-2: G. D. Hope (Fig. 190): Ed., "Quarterly Journal of Microscopical Science" (Fig. 191, 4-10): Société Mycologique de France (Fig. 197, 2-9): Dept. of Agric., Trinidad (Fig. 198, 2-5 & 7): Dir. Gen. Agric. Colon. Belg. (Fig. 204): Roy. Acad. Science, Amsterdam (Fig. 205, 2-11).

To the Director and staff of the Royal Botanic Gardens, Kew, especially the late Mr. G. Maase, Mr. A. D. Cotton and Miss E. Wakefield, I am under great obligations for the use of the rich collections and library while on leave, and for frequent assistance.

# BIBLIOGRAPHY.

The following does not pretend to be a complete list. In many cases only such references are given as will enable the full bibliography to be traced.

## TEXT BOOKS.

DE BARY: Comparative morphology and biology of the Fungi, Mycetozoa and Bacteria. ZOFF: Die Pilze. WARD: Disease in Plants. TUBEUF: Diseases of Plants induced by Cryptogamic parasites. MASSEE: Diseases of cultivated Plants and Trees. ERIKSSON: Fungoid diseases of agricultural Plants. DUGGAR: Fungous diseases of Plants. STEVENS & HALL: Diseases of economic Plants. COOK: Diseases of tropical Plants. PEILLIEUX: Maladies des Plantes agricoles. DELACROIX & MAUBLANC: Maladies des Plantes cultivées. IB: Maladies des Plantes cultivées dans les Pays Chauds. DUCOMET: Pathologie végétale. SORAUER: Handbuch der Pflanzenkrankheiten. KLEBAHN: Allgemeine Phytopathologie. KÜSTER: Pathologische Pflanzenanatomie. FERRARIS: I parassiti vegetali delle Piante coltivate od utili. ^

## CHAPTER I.

### THE NATURE OF FUNGI

*Rhizomorphs and Sclerotia*.—BOMMER: Solérotas et cordons mycéliens; Acad. R. So. Belg., Mem. Cour., LIV. PETCH: Termite Fungi; Ann. Peradeniya, V. *Reproduction*.—RAMSBOTTOM: Recent published results on the cytology of Fungus reproduction; Trans. Br. Myc. Soc., III *et seq.* *Dissemination*.—BUTLER: The dissemination of parasitic Fungi and international Legislation; Mem. Ag. Ind., Bot., IX, 1

## CHAPTER II.

### THE FOOD OF FUNGI.

LAFAR: Technical Mycology. IB: Handbuch der technischen Mykologie. LAURENT: Recherches expérimentales sur les maladies des Plantes; Ann. Inst. Past., XIII. MASSEE: On the origin of parasitism in Fungi; Phil. Trans. R. Soc., B, 197. IB: Observations on the study of Plant pathology; Journ. Ec. Biol., X. ORTON: The development of disease resistant varieties of Plants; IV Conf. Intern. Génét., p. 247. HEOKE: Beobachtungen der Ueberwinterungsart von Pflanzenparasiten; Naturw. Zeit. Land. u. Forstw., IX. TAUBENHAUS: A contribution to our knowledge of the morphology and life-history of *Puccinia malvacearum* Mont.; Phytopath., I. WARD: Recent researches on the parasitism of Fungi; Ann. Bot., XIX. MEHLER: Germination and infection with the fungus of the late blight of Potato (*Phytophthora infestans*); Wisc. Expt. Sta. Res. Bul. 37. DE BARY: Recherches sur le développement de quelques Champignons parasites; Ann. So. Nat., Ser. IV, XX. REED: Infection-experiments with *Erysiphe Cichoracearum*; Bul. Univ. Wisc., 250. RAY: Étude biologique sur le parasitisme: *Ustilago Maydis*; C. R., 136, p. 567. ZELLER: Studies in the physiology of the Fungi, II, *Lenzites aspiaria* Fr.; Ann. Mo. Bot. Gard., III. BUTLER: The

enzymes of *Polyporus squamosus*; Ann. Bot., XX. WARD: On the biology of *Stereum hirsutum*; Phil. Trans. R. Soc., B, 189. CLINTON: Chestnut bark disease; XXXVI Rep. Conn. Expt. Stat. GROOM: "Brown Oak" and its origin; Ann. Bot., XXIX. KLEBS: Physiologie der Fortpflanzung einiger Pilze; Prings. Jahrb., XXXII—XXXV. TROW: Observations on the biology and cytology of *Pythium ultimum*; Ann. Bot., XV. RAY: Variations des Champignons inférieurs sous l'influence du milieu; Thesis, Paris, 1897. PLANCON: Influence de divers milieux chimiques sur quelques Champignons du groupe des Dématées; Thesis, Paris, 1900. SCHIEMANN: Mutationen bei *Aspergillus niger*; Zeit. Induk. Abs. u. Vererbungsl., VIII. *Symbiosis*.—FREEMAN: The seed fungus of *Lolium temulentum*; Proc. R. Soc., 71. FUCHS: Beitrag zur Kenntnis des Loliumpilzes; Hedw., LI. RAYNER: Obligate symbiosis in *Calluna vulgaris*; Ann. Bot., XXIX.

### CHAPTER III.

#### LIFE-HISTORY OF PARASITIC FUNGI.

*Heterocism*.—KLEBAHN: Die Wirtswuchsolinden Rostpilze; 1904. *Specialisation of Parasitism*.—WARD: Recent researches, &c.; Ann. Bot., XIX. REED: Infection experiments with *Erysiphe Cichoracearum*; Bul. Univ. Wisc., 250. BUTLER & HAYMAN: Indian Wheat Rusts; Mem. Ag. Ind., Bot., I, 2. STAKMAN & PIEMRISEL: Biologic forms of *Puccinia graminis* on Cereals and Grasses; Jn. Ag. Res., X. REED: The Mildews of the Cereals; Bul. Tor. Bot. Club, XXXVI. NORTON: Methods used in breeding *Asparagus* for Rust-resistance; U. S. A. Dept. Ag., Bur. Pl. Ind., Bul. 263. STAKMAN: A study in Cereal Rusts: Physiological races; Minn. Expt. Stat. Bul. 138. ERIKSSON: Einige Studien ueber den Wurzelröster (*Rhizoctonia violacea*) der Möhre; Bakt. Centralb., 2nd Ab., X. POLE EVANS: South African Cereal Rusts; Jn. Ag. So., IV. VAVILOV: Immunity to Fungus diseases as a physiological test in genetics, &c.; Jn. Gen., IV. BARRIS: Variation of varieties of Beans in their susceptibility to Anthracnose; Phytopath., I. *Infection*.—WILTSHIRE: Infection and immunity studies on the Apple and Pear Scab Fungi, &c.; Ann. Appl. Biol., I. SALMON: Cultural experiments with the Barley Mildew, &c.; Ann. Myc., II. IS: On the stages of development reached by certain biologic forms of *Erysiphe*, &c.; New Phytol., IV. IS: Further cultural experiments with "Biologic Forms" of the *Erysiphaceae*; Ann. Bot., XIX. BALLS: Infection of Plants by Rust Fungi; New Phytol., IV. FROMME: Negative heliotropism of uredinospore germ-tubes; Amer. Jn. Bot., II. MIYOSHI: Die Durchbohrung von Membranen durch Pilzfäden; Prings. Jahrb., XXXIII. FULTON: Chemotropism of Fungi; Bot. Gaz., XLI. GIBSON: Notes on infection experiments with various Uredineae; New Phytol., III. STAKMAN: Relation between *Puccinia graminis* and Plants highly resistant to its attack; Jn. Ag. Res., IV. MARCHAL: Recherches biologiques sur une Chytridiée parasite du Lin; Rev. Myc., XXIII. GREGORY: Studies on *Plasmopara viticola*; Intern. Cong. Vitic., San Francisco, 1915. GÖTTER: Weitere Mittheilungen ueber den Krebs der Apfelbäume; Landw. Jahrb., IX. JONES, GRIDDINGS & LUTMAN: Investigations of the Potato Fungus, *Phytophthora infestans*; U. S. A. Dept. Ag., Bur. Pl. Ind., Bul. 245. ARNAUD & LAFONT: Accidents météorologiques et maladies du Murier; Ann. Éc. Nat. Ag. Montpellier, 2nd Ser., XI. BROWN: Studies in the physiology of parasitism, &c.; Ann. Bot., XXIX. KÜSTER: Die Gallen der Pflanzen. SALMON: Supplementary notes on the *Erysiphaceae*; Bul. Tor. Bot. Club, XXIX. POLE EVANS: Cereal Rusts; Ann. Bot., XXI. WARD: On the morphology of *Hemileia vastatrix*; Q. J. M. S., N. Ser., XXII. COLEMAN: Ueber *Sclerotinia trifoliorum*, &c.; Arb. K. Biol. Anst., V. MELHUS: Experiments on spore germination and infection in certain species of Oomycetes; Wisc. Expt. Stat., Res. Bul. 13. v. FÄHRER: Zur Infektion und Keimung der Uredosporen von *Hemileia vastatrix*; Ber. Deut. Bot.

Gezell, XXVIII. DUCOMET: Recherches sur le développement de quelques Champignons parasites à thalle subcuticulaire; Thèse, Paris, 1907. APPEL: Disease resistance in Plants; Science, N. S., 41. BURCK: Over de Koffiebladziekte &c.; Meded. 's Lands Plantentuin, V. VAN HALL: Robusta and some allied Coffee species; Ag. Bul. F. M. S., I.

## CHAPTER IV.

## THE CAUSATION OF DISEASE BY FUNGI.

*Symptoms.*—WARD: Disease in Plants. MOLLARD: Recherches sur les cécidies florales; Ann. Sc. Nat., Bot., 8th Ser., I. KÜSTER: Die Gallen der Pflanzen. SMITH: Crown gall studies, &c.; Jn. Ag. Sc., VI. HUTCHINSON: Rangpur Tobacco Wilt; Mem. Ag. Ind., Bact., I, 2. ILTIS: Ueber eine durch Maisbrand verursachte intracarpellare Prolifikation bei Zea Mays; Sitz. Kais. Akad. Wiss., Wien, CXIX. SYDOW & BUTLER: Fungi Indis Orientalis, II; Ann. Myc., V, p. 514. BABOLAY: Description of a new Fungus, *Æcidium esculentum*; Jn. Bom. Nat. Hist. Soc., V. ILTIS: Ueber einige bei Zea Mays L. beobachtete Atavismen, &c.; Zeit. Ind. Abst. u. Vererbungsl., V. EBERHARDT: Contribution à l'étude de *Cystopus candidus* Lév.; Bakt. Centralb., 2nd Ab., XII. PEM-LARY: Sur quelques effets de parasitisme de certains Champignons; Rev. Myc., XXI. *Morbid Anatomy.*—BEHRENS: Beiträge zur Kenntnis der Obstfäulnis; Bakt. Centralb., IV. STEVENS: Pathological histology of Strawberries affected by species of *Botrytis* and *Rhizopus*; Jn. Ag. Res., VI. DUCOMET: Recherches sur le brunissement des végétaux; Ann. Ec. Nat. Agric., Montpellier, XI. HAWKINS: Effect of certain species of *Fusarium* on the composition of the Potato tuber; Jn. Ag. Res., VI. TOBLER: Die Synchytrien; Archiv. für Protistenkunde, XXVIII. GUTTENBERG: Beiträge zur physiologischen Anatomie der Pilzgallen; Leipzig, 1905. DUCOMET: Recherches sur le développement de quelques Champignons parasites à thalle subcuticulaire; Thèse, Paris, 1907. WAKKER: Untersuchungen über der Einfluss parasitischer Pilze auf ihre Nährpflanzen; Prings. Jahrb., XXIV. COOK & TAUBENHAUS: The relation of parasitic Fungi to the contents of the cells of the host Plants; Del. Expt. Stat. Bul. 91 & 97. v.SCHREINK: Some diseases of New England Conifers; U. S. A. Dept. Ag., Div. Pat. & Phys., Bul. 25. Is: Two diseases of Red Cedar caused by *Polyporus juniperinus* and *Polyporus carneus*; ib. Bul. 21. Is: Diseases of deciduous forest Trees; U. S. A. Dept. Ag., Bur. Pl. Ind., Bul. 149. ZELLER: Studies in the physiology of the Fungi, II, *Lenzites sepiaria* Fr.; Ann. Mo. Bot. Gard., III. BULLER: The destruction of wood by Fungi; Science Progress, III. WEHMER: Die chemische Wirkung des Hausschwammes auf die Holzsubstanz; Ber. Deut. Bot. Gesell., XXXII. HAWKINS: The utilization of certain pentoses and compounds of pentoses by *Glomerella cingulata*; Am. Jn. Bot., II. LINDBRÖTH: Beiträge zur Kenntnis der Zersetzungserscheinungen des Birkenholzes; Naturw. Zeit. Land. u. Forstw., II. BROWN: Studies in the physiology of parasitism, I, the action of *Botrytis cinerea*; Ann. Bot., XXIX. HARTIG: Zersetzungserscheinungen des Holzes. BEHRENS: Untersuchungen ueber die Gewinnung der Hanffaser durch natürliche Röstmethoden; Bakt. Centralb., 2nd Ab., VIII. BULLER: The destruction of wooden paving blocks by the Fungus *Lenzites lepideus* Fr.; Jn. Ec. Bot., I. BIFFEN: On the biology of *Bulgaria polymorpha* Wett.; Ann. Bot., XV. HEDGECOCK & LONG: Preliminary notes on three rots of Juniper; Mycologia, IV. v.SCHREINK: A disease of the Black Locust (*Robinia pseudacacia* L.); Rep. Mo. Bot. Gard., XII. Is: Two trunk diseases of Mesquite; Ann. Mo. Bot. Gard., I. LONG: A honeycomb heart-rot of Oaks caused by *Stereum subulatum*; Jn. Ag. Res., V. WARD: On the biology of *Stereum hirsutum*; Phil. Trans. R. Soc., B, 189. v.SCHREINK: A trunk disease of the Lilac; Ann. Mo. Bot. Gard., I. SPATLING: The timber rot caused by *Lenzites sepiaria*; U. S. A. Dept. Ag., Bur. Pl. Ind., Bul. 214.

BUTLER: A study on Gummosis of *Prunus* and *Citrus*, &c.; *Ann. Bot.*, XXV. MARGIN: Sur le piétin ou maladie du pied du Blé; *Bul. Soc. Myc. Fr.*, XV. BROOKS: The development of *Gnomonia erythrostoma*, Pers.; *Ann. Bot.*, XXIV.

## CHAPTER V.

## PRINCIPLES OF THE CONTROL OF PLANT DISEASE.

MELHUS: Hibernation of *Phytophthora infestans* in the Irish Potato; *Jn. Ag. Res.*, V, p. 91. BROOKS & COOLEY: Temperature relations of Apple-rot Fungi; *Jn. Ag. Res.*, VIII. STEVENS: Rhizopus rot of Strawberries in transit; *U. S. A. Dept. Ag. Bul.* 531 (Prof. Paper). HARTER: Storage-rots of economic Aroids; *Jn. Ag. Res.*, VI. HUTCHINSON: Bacterial rot of stored Potato tubers; *Mem. Ag. Ind., Bact.*, I, 6. GÜSSOW: Smut diseases of cultivated Plants; *Cent. Expt. Farm, Ottawa, Bul.* 73. PRIDEAM: Flag Smut of Wheat; *Ag. Gaz.*, N. S. W., XXIV. BUTLER & HAYMAN: Indian Wheat Rusts; *Mem. Ag. Ind., Bot.*, I, 2. Report *Ag. Res. Inst., Pusa*, 1912-13. MASSEN: Some observations on the study of Plant Pathology; *Jn. Ec. Biol.*, X. DUGGAR: Early Blight of Celery; *Cornell Expt. Sta. Bul.* 132. IB: Fungus diseases of Plants, p. 68. SMITH: Asparagus and Asparagus Rust in California; *Cal. Expt. Sta. Bul.* 165. MUENCH: Untersuchungen ueber Immunität und Krankheitsempfanglichkeit der Holzpflanzen; *Naturw. Zeit. Land u. Forstw.*, VII. APPEL: The relation between scientific botany and phytopathology; *Ann. Mo. Bot. Gard.*, II. MÜLLER THURGAU: Die Edelfäule der Trauben; *Landw. Jahrb.*, 1888. BEHRENS: Beiträge zur Kenntnis der Obstfaulnis; *Bakt. Centralb.*, IV. LAURENT: Recherches expérimentales sur les maladies des Plantes; *Ann. Inst. Past.*, XIII. IB: Recherches de biologie expérimentale appliquée à l'agriculture, I. DE BARY: Ueber einige Sclerotinien und Sclerotienkrankheiten; *Bot. Zeit.*, XLIV. VIALA & PACOTTET: Sur la culture du Black-rot; *Rev. de Viticult.*, XXI. NOWELL: Rosellinia root diseases in the Lesser Antilles; *West Ind. Bul.*, XVI. Virulence.—BUTLER: The dissemination of parasitic Fungi, &c.; *Mem. Ag. Ind., Bot.*, IX, 1. COPELAND: Java and the Philippines; *Phil. Ag. & For.*, IV. WURTH: Degeneratie van Robustakoffie?; *Meded. Proefstat. Malang*, IV. Predisposition and Immunity.—ORTON: The development of disease resistant varieties of Plants; *IV Conf. Intern. Génét.*, 1911. FREEMAN: Resistance and immunity in Plant diseases; *Phytopath.*, I. BARRUS: An Anthracnose-resistant red Kidney Bean; *Phytopath.*, V. BIFFEN: Studies in the inheritance of disease resistance; *Jn. Ag. Sc.*, II. POLE EVANS: South African Cereal Rusts; *Jn. Ag. Sc.*, IV. BAKKE: Late Blight of Barley; *Proc. Iowa Acad. Sc.*, XIX. GILMAN: The relation of temperature to the infection of Cabbage by *Fusarium conglutinans*; *Phytopath.*, IV. IB: Cabbage Yellows and the relation of temperature to its occurrence; *Ann. Mo. Bot. Gard.*, III. BERGET: Résistance comparée de divers cépages vinifères au Rot-gris; *Rev. de Viticult.*, XIV. JONES, GIDDINGS & LUTMAN: Investigations of the Potato Fungus, *Phytophthora infestans*; *U. S. Dept. Ag., Bur. Pl. Ind.*, Bul. 245. v. FÄHRER: Die Krankheiten und Schädlinge des Kaffees; *Bakt. Centralb.*, XXI. LEWTON-BRAIN: Rind disease of the Sugar cane; *Expt. Stat. Hawaii Sugar Planters' Assn., Dn. Path. Phys.*, Bul. VII. BUTLER & HAFIS: Red rot of Sugar cane; *Mem. Ag. Ind., Bot.*, VI, 5. ORDNUNG: Immune Pflanzen; *Mitt. Deut. Dendro. Gesell.*, XXII. MORGENTHAU: Ueber die Bedingungen der Teleutosporenbildung bei der Uredineen; *Bakt. Centralb.*, 2nd Ab., XXVII. GÄSSNER: Untersuchungen ueber die Abhängigkeit des Auftretens der Getreideroste vom Entwicklungszustand der Nährpflanze; *Bakt. Centralb.*, 2nd Ab., XLIV. APPEL: Ueber bestandweises Absterben von Roterlen; *Naturw. Zeit. Land u. Forstw.*, II. BUIS: Notes sur les maladies des Caféiers, &c.; *L'Agric. Prat. Pays Chauds*, VI. MELHUS: Germination and infection with the Fungus of the Late Blight of Potato; *Wisc. Expt. Sta. Res. Bul.* 37. APPEL: Disease resistance in Plants; *Science*, N. S., 41. NILSSON EHLE: Kreuzungsunter-

suchungen an Hafer und Weizen, II; Lunds Univ. Arsk., 2nd Af., VII. BLAIRINGHEM: Sur le propagation des Rouilles de Céréales en Suède et en France; Bul. Soc. Bot. Fr., LXI. HOWARD & HOWARD: Note on immune Wheat; Jn. Ag. Sc., II. SOMERVILLE: Report on the enquiry conducted by the Society into the disease of the Larch; Trans. Eng. Arbor. Soc., II. FREEMAN: Resistance and immunity in Plant diseases; Phytopath., I. VALLEAU: Varietal resistance of Plums to Brown-rot; Jn. Ag. Res., V. LANG: Zum Parasitismus der Brandpilze; Jahr. Verein. Angew. Bot., X. DE BARY: Ueber einige Sclerotinien und Sclerotienkrankheiten; Bot. Zeit., XLIV. STAKMAN: Relation between Puccinia graminis and Plants highly resistant to its attack; Jn. Ag. Res., IV. IS. & PIEMEISEL: Biologic forms of Puccinia graminis on Cereals and Grasses; *ib.*, X. WILTSHIRE: Infection and immunity studies on the Apple and Pear Scab Fungi; Ann. Appl. Biol., I. VIALA & PACOTTET: Sur la culture du Black rot; C. R., 138. REDDICK: The Black rot disease of Grapes; Cornell Expt. Sta. Bul. 293. COMES: Della resistenza dei Frumenti alle Ruggini; Ann. R. Scuola Agric., Portici, XII. COOK & TAUBENHAUS: The relation of parasitic Fungi to the contents of the coils of the host Plants; Del. Expt. Sta. Bul. 91 & 97. BUTLER: Fungous diseases of Sugarcane in Bengal; Mem. Ag. Ind., Bot., I, 3. COUDEAC: IV Conf. Intern. Génét.; Paris, 1911, p. 264. DELACROIX: Observations et recherches au sujet de la "Pourriture grise," &c.; Bul. Mens. Min. Agric., April, 1905. SALMON: American Gooseberry Mildew; Jn. Bd. Ag., XX. JONES, GIDDINGS, & LUTMAN: Investigations of the Potato Fungus, Phytophthora infestans; U. S. A. Dept. Ag., Bur. Pl. Ind., Bul. 245. PAULSEN: Kartoffelkultur und Erträge; Biederm. Centralb., XVI. SPINKS: Factors affecting susceptibility to disease in Plants; Jn. Ag. Sc., V. RUSSELL: Fertilisers and Manures, 1915. BRIGGS: The field treatment of Tobacco Root-rot; U. S. A. Dept. Ag., Bur. Pl. Ind., Circ. 7. LAURENT: Recherches expérimentales sur les maladies des Plantes; Ann. Inst. Past., XIII. IS: Recherches de biologie expérimentale appliquée à l'agriculture, I. MARCHAL: *ib.* and C. R., 135, p. 1067. RUSSELL: Soil conditions and Plant growth. IS: Soils and Manures, 1915. SACCARDO & BERLESE: Una nuova malattia del Frumento; Riv. Pat. Veg., IV. RAMSBOTTOM: Iris Leaf-blotch; Jn. R. Hort. Soc., XL. REED: Infection experiments with Erysiphe Cichoracearum; Bul. Univ. Wisc. 250. HEWITT: Rose Mildew; Phytopath., III. FREEMAN: Timothy Rust in the United States; U. S. A. Dept. Ag., Bur. Pl. Ind., Bul. 224. SALMON: Cultural experiments with "Biologic forms" of the Erysiphaceae; Phil. Trans. R. Soc., B. 197. IS: Cultural experiments with the Barley Mildew; Ann. Myc., II. IS: Cultural experiments with an Oidium on Euonymus japonicus; Ann. Myc., III. Deterioration.—SUTTON: The alleged deterioration of Potatoes; Gard. Chron., 3rd Ser., XXXVIII. BUTLER: Tikka disease and the introduction of exotic Groundnuts in Bombay Presidency; Ag. Jn. Ind., IX. Actes V Congr. Intern. Riziculture, 1914. Epidemics.—MORELAND: in BUTLER & HAYMAN: Indian Wheat Rusts; Mem. Agr. Ind., Bot., I, 2. DASTUR: Conditions influencing the distribution of Potato Blight in India; Ag. Jn. Ind., XII. Methods of direct control.—LODEMAN: The spraying of Plants. BOURCART: Insecticides, Fungicides, and Weedkillers. ANDREWS & TUNSTALL: Notes on the spraying of Tea; Ind. Tea Ass. Bul. 1, 1915. ANSTEAD: The treatment of Fungoid diseases on Estates; Ag. Jn. Ind., XIII. Ann. Rep. Upper Shillong Ag. Stat., Assam, 1906. COLEMAN: Diseases of the Areca palm, I, Koleroga; Dept. Ag. Mysore, Mycol. Ser., Bul. II. STEWART: Botanical investigations; XXVI Ann. Rep. New York Expt. Stat. ANNETT & KAR: Amount of copper in Tea sprayed with Bordeaux mixture; Jn. Ag. Sc., III. DUGGAR & COOLEY: The effect of surface films and dusts on the rate of transpiration; Ann. Mo. Bot. Gard., I. BRENCHEY: Inorganic Plant poisons and stimulants, 1914. BUTLER: Bordeaux mixture; Phytopath., IV. SALMON: Making and application of Bordeaux mixture; Jn. Bd. Ag., XVI. BARKER & GIMINGHAM: The action of Bordeaux mixture on Plants; Ann. Appl. Biol., I. PETHYBRIDGE: Investigations on Potato diseases; Jn. Dept. Ag. & Tech. Ins. Ireland, X et seq. BUTLER: The Cuprammonium washes, &c.; Phytopath., VII. RAMSAY: Lime-sulphur sprays, &c.; Jn. Ag. Sc., VI. IS: Dept. Ag. N. S. W., Sc. Bul. 3. MUNN: Lime



sulphur w. Bordeaux mixture as a spray for Potatoes; New York Expt. Stat. Bul. 352; SALMON: American Gooseberry Mildew; Jn. Bd. Ag. XX. KRITZ: Peach Scab and its control; U. S. A. Dept. Ag. Bul. 395 (Prof. Paper). EYRE & SALMON: The fungicidal properties of certain spray fluids; Jn. Ag. Sc., VII.

## CHAPTER VI.

## CEREALS.

**General.**—RIEHL: Zusammenfassende Uebersichten; Bakt. Centralb., 2nd Ab., XXX *et seq.* ERIKSSON & HENNING: Die Getreideroste. SYDOW: Monographia Uredinearum. MCALPINE: The Rusts of Australia. 1b: The Smuts of Australia. 1ro: On the Uredinæ parasitic on the Japanese Graminæ; Jn. Sapp. Ag. Coll., III. POLE EVANS: Cereal Rusts; Ann. Bot., XXI. 1b: South African Cereal Rusts; Jn. Ag. Sc., IV. GASSNER: Untersuchungen ueber die Abhängigkeit des Auftretens der Getreideroste vom Entwicklungszustand der Nährpflanzen; Bakt. Centralb., 2nd Ab., XLIV. GÜSSOW: Smut diseases of cultivated Plants; Contr. Expt. Farm, Ottawa, Bul. 73. CLINTON: North American Ustilaginæ; Proc. Bost. Soc. Nat. Hist., XXXI, 9. 1b: Ustilaginæ; N. A. Flora, VII. BRIEFELD: Untersuchungen; V, XI, XII, XIII, XV. LANG: Parasitismus der Brandpilze; Jahrb. Ver. Angew. Bot., X. **Wheat.** *Rust.*—BUTLER & HAYMAN: Indian Wheat Rusts; Mem. Ag. Ind., Bot. I, 2. Ann. Rep. Cawnpore Agric. Stat., 1907. STAKMAN & PIEMISEL: Biologic forms of *Puccinia graminis* on Cereals and Grasses; Jn. Ag. Res., X. BAUDYS: Ein Beitrag zur Ueberwinterung der Rostpilze durch Uredo; Ann. Myc., XI. BEAUVERIE: Sur l'efficacité des germes de Rouilles, &c.; C. R., 158. BLARINGHEM: Sur la transmission héréditaire de la Rouille chez la Rose tremière; C. R., 157. 1b: Sur la propagation de Rouilles; Bul. Soc. Bot. Fr., LXI. *Smut.*—HEALD & WOOLMAN: Bunt or Stinking Smut of Wheat; Wash. Expt. Stat. Bul. 126. FAMINCYN: *Ref. Bot. Centralb.*, 122, p. 319. PRIDHAM: Flag Smut of Wheat; Ag. Gaz., N. S. W., XXIV. *Mildew.*—SALMON: A Monograph of the Erysiphacæ; Mem. Tor. Bot. Club, IX. REED: The Mildews of the Cereals; Bul. Tor. Bot. Club, XXXVI. FOËX: Les conidiophores des Erysiphacées; Ann. Éc. Nat. Agric. Montpellier, N. Ser., XI. SALMON: Notes on some species of Erysiphacæ from India; Ann. Myc., V. GAROVAGLIO & CATTANEO: Sulla Erysiphe graminis e sulla Septoria Tritici, &c.; Arch. Lab. Bot. Critto. Pavia, II—III. *Leaf Spot.*—MANGIN: Sur le Septoria gramineum Desm., &c.; Bul. Soc. Myc. Fr., XV. VOGLINO: Ricerche intorno allo sviluppo ed al parasitismo della Septoria gramineum, &c.; Atti R. Acca. di Agric., Torino, XLVI. *Mould.*—BANCROFT: Researches on the life-history of parasitic Fungi, I, Cladosporium herbarum Lk.; Ann. Bot., XXIV. BARBER: Diseases of Andropogon Sorghum in the Madras Presidency; Dept. Land Rec. and Ag., Madras, Bul. II, 49. **Oats.** *Smut.*—(See under *General*). *Rust.*—(See under *General and Wheat Rust*). *Leaf Spot.*—RAVN: Nogle Helminthosporium-Arter, &c.; Bot. Tidssk., XXIII. **Barley.** *Smut.*—(See under *General*). *Rust.*—(See under *General and Wheat Smut*). *Late Blight and Stripe Disease.*—RAVN: I. c. BAKKE: Late Blight of Barley; Proc. Iowa Acad. Sc., XIX. JOHNSON: The ascigerous stage of Helminthosporium teres Sacc., Phytopath., IV. MÜLLER & MOLZ: Versuche zur Bekämpfung der durch Pleospora trichostoma hervorgerufenen Streifenkrankheit der Gerste; Deutsch. Landw. Presse, XLI. **Maize.** *Downy Mildew.*—BUTLER: The Downy Mildew of Maize; Mem. Ag. Ind., Bot., V, 5. RUTGERS: De Peronosporaziekte der Mais; Meded. Labor. Plantenziek., Buitenzorg, 22. *Rust.*—KLEPAHN: Kryptogamenflora der Mark Brandenburg, V, a, 3, p. 466. POLE EVANS: Rep. of Plant Pathologist and Mycologist in Ann. Rep. S. Afr. Dept. Ag., 1911. *Smut.*—PIEMISEL: Factors affecting the parasitism of Ustilago Zeæ; Phytopath., VII. GUTTENBERG: Beiträge zur physiologischen Anatomie der Pilzgallen, 1905. CHIFFLOT:

Castration thelygène chez *Zea Mays* var. *tunicata* produite par l'*Ustilago Maydis*; C. R., 148. **ILTZS**: Ueber eine durch Maisbrand verursachte intracarpellare Prolifikation, &c.; Sitz. K. Akad. Wiss., Wien, CXIX. **Head Smut**.—**POTTER**: Head Smut of Sorghum and Maize; Jn. Ag. Res., II. **Leaf Blight**.—**CHESTER & SMITH**: Notes on Fungous diseases in Delaware; Del. Expt. Stat. Bul. 63. **ROBINSON**: Corn Leaf Blight in the Philippines; Phil. Ag. Rev., IV. **POLE EVANS**: Rep. of Plant Pathologist and Mycologist in Ann. Rep. S. Afr. Dept. Ag., 1912-13. **ANON**: Blight in Maize; Ag. Gaz. N. S. W., XXVI, p. 388. **Jowar. Downy Mildew**.—**KULKARNI**: Observations on the Downy Mildew of Bajra and Jowar; Mem. Ag. Ind., Bot., V, 5. **Rust**.—**WATT**: Indian Fungi; Agric. Ledger, 1895, No. 20. **BARBER**: Diseases of Andropogon Sorghum in the Madras Presidency; Dept. Land Rec. and Ag., Madras, Bul. II, 49. **BUSSE**: Ueber den Rost der Sorghum-Hirse in Deutsch Ost-Afrika; Ber. Deut. Bot. Gesell., XX. **Smuts**.—**KULKARNI**: Smuts of Jowar (Sorghum) in the Bombay Presidency; Bul. Ag. Res. Inst., Pusa, 78. **BUSSE**: Untersuchungen ueber die Krankheiten der Sorghumhirse; Arb. K. Biol. Anat., IV. **POLE EVANS**: Smut in Kaffir Corn; Ag. Jn. Un. S. Afr., VII. **POTTER**: The Loose kernel Smut of Sorghum; Phytopath., V. **Red Leaf Spot**.—**WILSON**: The identity of the Anthracnose of Grasses in the United States; Phytopath., IV. **Bajra. Green Ear Disease**.—**BUTLER**: Some diseases of Cereals caused by *Sclerospora graminicola*; Mem. Ag. Ind., Bot., II, 1. **KULKARNI**: Observations on the Downy Mildew of Bajri and Jowar; Mem. Ag. Ind., Bot., V, 5. **YAMADA**: On the occurrence of *Sclerospora macrospora* Sacc. on Rice; Marioka Ag. and Forest High School, 1912 (Japanese). **Rust**.—**ZIMMERMANN**: Untersuchungen ueber tropische Pflanzenkrankheiten; Ber. Land. u. Forstw. Deut.-Ostafrika, II. **Rice. General**.—**BUTLER**: Some diseases of Rice; Bul. Ag. Res. Inst., Pusa, 34. **FULTON**: Diseases affecting Rice in Louisiana; Lo. Expt. Stat. Bul. 105. **MIYAKE**: Studien ueber die Pilze der Reis-pflanze in Japan; Jn. Coll. Ag. Imp. Uni., Tokyo, II. **Smut**.—**RUTGERS**: Stufbrand bij Rijet (*Tilletia horrida* Tak.); Meded. Labor. Plantenziek., Buitenzorg, No. 11. **False Smut**.—**HUTCHINSON**: Bakhar—The Indian Rice beer ferment; Mem. Ag. Ind., Bact., I, 6. **Sclerotial Disease**.—**SHAW**: A sclerotial disease of Rice; Mem. Ag. Ind., Bot., VI, 2. **SAWADA**: Hypochynus on some cultivated Plants in Formosa; Bot. Mag., Tokyo, XXVI. **Smaller Millets**. **BUTLER**: Some diseases of Cereals caused by *Sclerospora graminicola*; Mem. Ag. Ind., Bot., II, 1. **BREFELD**: Untersuchungen, V, XII, & XIII. **HROKE**: Belzversuche gegen Hirsenbrand; Zeit. Landw. Versuchsw. Oesterr. VI. **HORI**: Seed infection by Smut Fungi of Cereals; Bul. Centr. Ag. Expt. Stat., Tokyo, I. **SYDOW**: *Novae fungorum species*, VI; Ann. Myc., IX. **RACIBORSKI**: *Ustilago Coicis* Bref.; Parasit. Algen u. Pilze Javas, III.

## CHAPTER VII.

### PULSE CROPS.

**General**.—**BERLESE**: Saggio di una monografia delle Peronosporacee; Riv. Pat. Veg., VI, VII, IX. **SYDOW**: Monographia Uredinearum. **McALPINE**: Rusts of Australia. **Pigeon pea. Wilt**.—**BUTLER**: The Wilt disease of Pigeon Pea, &c.; Mem. Ag. Ind., Bot., II, 9. **WOLLENWEBER**: Pilzparasitäre Welkekrankheiten der Kulturpflanzen; Ber. Deut. Bot. Gesell., XXXI. **Peas. Downy Mildew**.—**BRION**: Malattie delle Veccie; Atti Ist. Bot. Pavia, 2nd Ser., XIV, p. 416. **FRANK**: Kampfbuch gegen die Schädlinge unserer Feldfrüchte, 1897, p. 228. **Powdery Mildew**.—**VAN HOOK**: Blighting of field and garden Peas; Ohio Expt. Stat. Bul. 173. **SALMON**: Supplementary notes on the Erysiphaceae; Bul. Tor. Bot. Club, XXIX. **Rust**.—**JORDI**: Beiträge zur Kenntnis der Papilionaceen bewohnenden Uromyces-Arten; Rakt. Centralb., 2nd Ab., XI. **Beans. General**.—**WINTERL**: Diseases of Beans; Corn. Univ. Expt. Stat. Bul. 239. **HALSTED**: Bean diseases and their remedies; N. J. Expt. Stat. Bul. 151. **Anthrax**.—**EDGERTON**: Effect of temperature on *Glemorella*; Phytopath., V. **SERIES**

& WOOD: Studies of Fungous parasites belonging to the genus *Glomerella*; U. S. A. Dept. Ag., Bur. Pl. Ind., Bul. 332. BARRUS: An Anthracnose-resistant red Kidney Bean; *Phytopath.*, V. *Rust*.—LACON: Ueber die Empfänglichkeit von *Phaseolus vulgaris* L. and *Ph. multiflorus* Willd. für der Bohnenrost, &c.; *Zeit. Pflanzenk.*, XXVI. GASSNER: Algunas observaciones sobre el "Polvillo" de los Potoros (*Uromyces appendiculatus*); *Rev. Sec. Agron.*, Montevideo, IV. ORTON: The development of disease resistant varieties of Plants; IV Conf. Intern. Génét., 1911. *Leaf Spot*.—JACKSON: Diseases of field crops in Delaware; *Dela. Expt. Stat. Bul.* 83. RACIBORSKI: Pflanzen-pathologische aus Java; *Zeit. Pflanzenk.*, I. *Oowpea*. *Die Back*.—So. Rep. Ag. Res. Inst., Pusa, 1916-17, p. 63. *Root Rot*.—SHAW: The morphology and parasitism of *Rhizoctonia*; *Mem. Ag. Ind., Bot.*, IV, 6. DUGGAR: *Rhizoctonia Crocorum* (Pers.) DC. & R. Solani Kühn, &c.; *Ann. Mo. Bot. Gard*, II. PELTIER: Parasitic *Rhizoctonias* in America; III. *Expt. Stat. Bul.* 189. PETHYBRIDGE: Investigations on Potato diseases; *Jn. Dept. Ag. & Tech. Instr. Ireland*, XV—XVI. *Soy Bean*. *Downy Mildew*.—SYDOW & BUTLER: *Fungi Indis Orientalis*, IV; *Ann. Myc.*, X. NAUMOFF: *Fungi Ussuriensis*; *Bul. Soc. Myc. Fr.*, XXX. Parasitic fungi of Formosa; *Bot. Mag.*, Tokyo, XXVIII, p. (423). *Khesari*. *Rust*.—BARCLAY: On some Rusts and Mildews in India; *Jn. Bot.*, XXVIII. *Gram*. *Blight*.—STONE: The life history of *Ascochyta* on some leguminous Plants; *Ann. Myc.*, X. *Rust*.—BARCLAY: Some Rusts and Mildews in India; *Jn. Bot.*, XXVIII. BOYER & JACZEWSKI: Matériaux pour la flore Mycologique des environs de Montpellier; *Ann. Éc. Nat. Agric. Montp.*, 1894. *Guar*. *Mildew*.—SALMON: On *Oidiopsis taurica* (Lév); *Ann. Bot.*, XX. FÖRER: Recherches sur *Oidiopsis taurica*; *Bul. Soc. Myc. Fr.*, XXIX.

## CHAPTER VIII.

## VEGETABLES, ROOT-CROPS, AND OIL-SEEDS.

**Potato.** *General*.—BUTLER: Potato diseases of India; *Agric. Ledger*, 1903, No. 4. McALPINE: Potato diseases in Australia; Dept. of Ag., Victoria. PETHYBRIDGE: Investigations on Potato diseases; *Jn. Dept. Ag. & Tech. Instr. Ireland*, X—XVII. *Bacterial Wilt*.—COLEMAN: The Ring disease of Potatoes; Dept. Ag., Mysore, *Mycol. Ser.*, Bul. 1. SMITH: Bacteria in relation to Plant diseases, III. *Blight*.—BRECKLEY: Observations on the Potato murrain; *Jn. R. Hort. Soc.*, I. MARTIUS: Ueber die diesjährige Krankheit der Kartoffeln oder die nasse Fäule, 1845. LE CORNU: The Potato in Jersey; *Jn. R. Agric. Soc. Eng.*, 1870. JONES, GIDDINGS, & LUTMAN: Investigations of the Potato Fungus, *Phytophthora infestans*; U. S. A. Dept. Ag., Bur. Pl. Ind., Bul. 245. DASTUR: The Potato Blight in India; *Mem. Ag. Ind., Bot.*, VII, 3. IS: Conditions influencing the distribution of Potato Blight in India; *Ag. Jn. Ind.*, XII. MELHUS: Hibernation of *Phytophthora infestans* in the Irish Potato; *Jn. Ag. Res.*, V. ROSENBAUM: Studies of the genus *Phytophthora*; *Jn. Ag. Res.*, VIII. Lounsbury quoted by McAlpine, l. c., p. 17. STEWART, FRENCH, & SERRINE: Potato spraying experiments 1902-1911; *Geneva N. Y. Expt. Stat. Bul.* 349. BUTLER: Spraying Potatoes; *Ag. Jn. Ind.*, II, p. 95. *Leaf Blotch*.—LAGERHEIM & WAGNER: Bladsflekksjuka a Potatis; K. Landtbr. Akad. Handl., XLII. DUOMET: Sur quelques maladies des Plantes cultivées; *Ann. Éc. Nat. Agric. Rennes*, IV. JONES & POMEROY: The Leaf-blotch disease of Potato; *Rep. Vermont Expt. Stat.*, 1906. *Early Blight*.—JONES & GROUT: Notes on two species of *Alternaria*; *Bul. Tor. Bot. Club*, XXIV. JONES: Certain Potato diseases and their remedies; *Vermont Expt. Stat.*, Bul. 72. MASSEE: Perpetuation of Potato disease and Potato Leaf curl by means of hibernating mycelium; *Kew Bul.*, 1906. COCKAYNE: The facultative saprophytism of *Alternaria Solani*; *New Zealand Dept. Ag., Dn. Biol. Tech. Paper* I. *Root and Tuber Rot*.—SHAW & AJREKAR: The genus *Rhizoctonia* in India; *Mem. Ag. Ind., Bot.*, VII, 4. **BRASSICAS.**

*White Rust*.—**EEBERHARDT**: Contribution à l'étude de *Cystopus candidus* Lév.; Bakt. Centralb., 2nd Ab., XII. **MOLLARD**: Recherches sur les oécidies florales; Ann. So. Nat., Bot., 8th Ser., I. **MELIUS**: Experiments on spore germination and infection in certain species of Oomycetes; Wisc. Expt. Stat. Bul. 15. **WAGER**: On the structure and reproduction of *Cystopus candidus*; Ann. Bot., X. *Downy Mildew*.—**EEBERHARDT**: l. c. **WAGER**: On the fertilization of *Peronospora parasitica*; Ann. Bot. XIV. **TUBERV**: Kranke Rettiche; Naturw. Zeit. Land. u. Forstw., VI. v. **MANNA GETTA**: Ueber eine neue Krankheit unserer Radieschen; Lotos, XIX. v. **SCHRENK**: On the occurrence of *Peronospora parasitica* on Cauliflower; XVI Ann. Rep. Mo. Bot. Gard. **COMES**: Notizie intorno ad alcune Crittogame parassite delle Piante agrarie; Ann. R. Scuol. Agr., Portici, II. **MAGNUS**: Einige Bemerkungen ueber die auf *Phalaris arundinacea* auftretenden Puccinien; Hedw., XXXIII. *Powdery Mildew*.—**SALMON**: Monograph of the Erysiphaceæ; Mem. Tor. Bot. Club, IX. *Blight*.—**KUEHN**: Die Krankheiten der Kulturgewächse. **MASSEE**: Fungi exotici, III; Kew Bul., 1901. *Tomato. Blight*.—**KERN & ORTON**: *Phytophthora infestans* on Tomatoes; Phytopath., VI. *Fruit Rot*.—**MASSEE**: On the presence of hibernating mycelium of *Maclosporium Solani* Ck. in Tomato seed; Kew Bul., 1914. **POOL**: Some Tomato fruit rot during 1907; XXI Ann. Rep. Nebr. Expt. Stat. **SHEAR & WOOD**: Studies of Fungous parasites belonging to the genus *Glomerella*; U. S. A. Dept. Ag., Bur. Pl. Ind., Bul. 252. **KRUEGER**: Beiträge zur Kenntnis einiger Gloeosporien; Arb. K. Biol. Anat., IX. **TAUBENHAUS**: A further study of some Gloeosporiums and their relation to a Sweet-potato disease; Phytopath., II. *Wilt*.—**MASSEE**: The Sleeping disease of Tomatoes; Jn. R. Hort. Soc., XIX. **SMITH**: Tomato diseases in California; Cal. Expt. Stat. Bul. 175. **WOLLENWEBER**: Studies on the Fusarium problem; Phytopath., III. *Is. Pilzparasitäre Welkekrankheiten der Kulturpflanzen*; Ber. Deut. Bot. Gesell., XXXI. **HUMPHREY**: Studies on the relation of certain species of *Fusarium* to the Tomato blight of the Pacific north-west; Wash. Expt. Stat. Bul. 115. *Brinjal. Fruit Rot*.—(See under *Tomato*). *Bhindi. Mildew*.—**SALMON**: Supplementary notes on the Erysiphaceæ; Bul. Tor. Bot. Club, XXIX. **BUBAK**: Einige interessante Pflanzenkrankheiten aus Bulgarien; Bakt. Centralb., 2nd Ab., XXXI. *Colocasia. Blight*.—**BUTLER & KULKARNI**: Studies in Peronosporaceæ: *Colocasia* blight caused by *Phytophthora Colocasiae* Rac.; Mem. Ag. Ind., Bot., V, 5. **MENDIOLA & ESPINO**: Some Phycomycetous diseases of cultivated Plants in the Philippines; Phil. Ag. and For., V. **ROSENBAUM**: Studies of the genus *Phytophthora*; Jn. Ag. Res., VIII. *Cassava. Leaf Spot*.—**ZIMMERMANN**: Untersuchungen ueber tropische Pflanzenkrankheiten; Ber. Land. u. Forstw. Deut. Ostafrika, II. **ROBER**: Ann. Rep. of Mycologist; Bd. of Ag. Trinidad, 1910, p. 7. *Is*: Fungous diseases of Cassava; Bul. Dept. Ag. Trin. and Tob., XIV. *Cucurbits. Downy Mildew*.—**STONE**: Downy Mildew of Cucumbers; Mass. Expt. Stat. Circ. 40. **WILSON**: Studies in North American Peronosporales, II; Bul. Tor. Bot. Club, XXXIV. **ZIMMERMANN**: l. c. **CARRUTHERS**: Ann. Rep. Govt. Mycologist; Circ. Roy. Bot. Gard., Ceylon, II, 1. **KOCK**: Die Resultate der Versuche des Jahres 1908 zur Bekämpfung des falschen Meltauens der Gurken; Zeit. Land. Versuchsw. Oester., XII. *Powdery Mildew*.—**HUMPHREY**: Powdery Mildew of the Cucumber; IX & X Rep. Mass. Expt. Stat. **REED**: Infection experiments with *Erysiphe Cichoracearum*; Wisc. Expt. Stat. Bul. 250. **SALMON**: Monograph of the Erysiphaceæ; Mem. Tor. Bot. Club, IX. *Root Rot*.—**SEAW & AJREKAR**: The genus *Rhizoctonia* in India; Mem. Ag. Ind., Bot., VII, 4. *Celery. Leaf Spot*.—**DUGGAR**: Two destructive Celery Blights; Cornell Expt. Stat. Bul. 132. **STURGIS**: On the prevention of Leaf-blight and Leaf-spot of Celery; XXI Rep. Conn. Expt. Stat. **KLEBAHN**: Krankheiten des Sellerie; Zeit. Pflanzenk., XX. *Amaranthus. White Rust*.—**SYDOW & BUTLER**: Fungi Indis Orientalis, II; Ann. Myc., V, p. 514. **MASALONGO**: Sull' alterazione di colore dei fiori dell'*Amaranthus retroflexus* infestati dalle oospore di *Cystopus Bliti*; Nuov. Giorn. Bot. Ital., XXIII. *Is*: Di un nuovo micococcidio dell'*Amaranthus silvestris*; Bol. Soc. Bot. Ital., 1904. **WILSON**: Studies in North American Peronosporales, I; Bul. Tor. Bot. Club, XXXIV. *Onopodiūm. Downy Mildew*.—**WILSON**:

*ib*, VI; Mycologia, VI. NAOUMOFF: Fungi Ussuriensis; Bul. Soc. Myc. Fr., XXX. **Portulaca. White Rust.**—WILSON: *ib*, I; Bul. Tor. Bot. Club, XXXIV. **Groundnut. Tikka.**—BUTLER: Tikka disease and the introduction of exotic Groundnuts in the Bombay Presidency; Ag. Jn. Ind., IX. WOLF: Further studies on Peanut Leaf Spot; Jn. Ag. Res., V. **Root Rot.**—SHAW & AJREKAR: The genus *Rhizoctonia* in India; Mem. Ag. Ind., Bot., VII, 4. **Linseed. Rust.**—ARTHUR: Cultures of Uredines in 1906; Jn. Mycol., XIII. FROMME: Sexual fusions in Flax Rust; Bul. Tor. Bot. Club, XXXIX. McALPINE: Rusts of Australia, 1906. WATT: Indian Fungi; Agric. Ledger, 1895, No. 20, p. 18. BUCHHEIM: Zur Biologie von *Melampsora lini*; Ber. Deut. Bot. Gesell., XXXIII. **Castor. Seedling Blight.**—DASTUR: On *Phytophthora parasitica*, &c.; Mem. Ag. Ind., Bot., V, 4. ROSENBAUM: Studies of the genus *Phytophthora*; Jn. Ag. Res., VIII. **Rust.**—AJREKAR: The Castor Rust (? *Melampsorella Ricini* de Ton.); Jn. Bom. Nat. Hist. Soc., XXI.

## CHAPTER IX.

## DYE, DRUG, AND SPICE CROPS.

**Tobacco. Bacterial Wilt.**—HUTCHINSON: Rangpur Tobacco Wilt; Mem. Ag. Ind., Bact., I, 2. SMITH: Bacteria in relation to Plant diseases, III. HONING: Onderzoekingen over de virulentie van *B. solanacearum*, &c; Bul. Deli Proefstat., II. GARNER, WOLF, & MOSS: The control of Tobacco Wilt in the flue-cured district; U. S. A. Dept. Ag. Bul. 562 (Prof. Paper). **Mildew.**—SALMON: Monograph of the *Erysipheae*; Mem. Tor. Bot. Club, IX, p. 204. COMES: Notizie intorno ad alcune *Crittogame* parassite delle Piante agrarie; Ann. R. Scuola Agric. Portici, II. PETERS & SCHWAB: Krankheiten und Beschädigungen des Tabaks; Mitt. K. Biol. Anst., 13. PETCH: Diseases of Tobacco in Dumbura; Circ. R. Bot. Gard', Ceylon, IV, 7. MORSTATT: Beobachtungen ueber das Auftreten von Pflanzenkrankheiten im Jahre 1911; Der Pflanze VIII. MONSON: Ann. Rep. Adviser for Tobacco; Ann. Rep. Dept. Ag. Br. E. Afr. 1913 to 1916. **Leaf Spot.**—STROUBIS: On a destructive disease of Tobacco in S. Carolina. XX Rep. Conn. Expt. Stat. JENSEN: Versuche mit drei Tabakskrankheiten; Jaarboek Dept. Landb. Neerl. Indie, 1908. BAKER: A review of some Philippine Plant diseases Phil. Ag. and For., III. **Mosaic.**—ALLARD: Review of investigations on Mosaic disease of Tobacco; Bul. Tor. Bot. Club, XLI. 1b: Effect of dilution upon the infectivity of the virus of the Mosaic disease of Tobacco; Jn. Ag. Res., III. 1b: A specific Mosaic disease in *Nicotiana glauca*, &c.; Jn. Ag. Res., VII. **Opium poppy. Downy Mildew.**—Blight in the Poppy crop in the Behar and Benares Agencies; Sel. Records Finance Dept., Ind., Papers, 1871. CUNNINGHAM: On certain diseases of fungal and algal origin, &c.; So. Mem. Med. Off. Army of Ind., X. MAGNUS: Die Peronosporosen der Provinz Brandenburg; Verh. Bot. Ver. Prov. Brand., XXXV. WILSON: Studies in North American Peronosporales, III; Bul. Tor. Bot. Club, XXXV. BERLESE: Saggio di una monografia delle Peronosporacee; Riv. Pat. Veg., X. **Turmeric. Leaf Spot.**—BUTLER: The Leaf Spot of Turmeric (*Taphrina maculans* sp. nov.); Ann. Myc., IX. SYDOW: Zweiter Beitrag zur Kenntnis der parasitischen Pilzflora des nördlichen Japans; Ann. Myc., XII. **Ginger. Soft Rot.**—McRAE: Soft Rot of Ginger in the Rangpur District, Eastern Bengal; Ag. Jn. Ind., VI. Sc. Rep. Ag. Res. Inst., Pusa, 1916-17, p. 67. **Ohili. Die Back.**—SYDOW: Beiträge zur Kenntnis der Pilzflora des südlichen Ostindiens, I; Ann. Myc., XI. BAKER: The lower Fungi of the Philippine Islands, 1st Suppl. Leaflets Phil. Bot., VII, p. 2513. Sc. Rep. Ag. Res. Inst., Pusa, 1916-17, p. 63. **Ripe Rot.**—SHEAR & WOOD: Ascogenous forms of *Gloeosporium* and *Colletotrichum*; Bot. Gaz., XLIII. HALSTED: A study of Solanaceous Anthracnoses; Bul. Tor. Bot. Club, XX. **Anthracnose.**—TAUBENHAUS: A further study of some *Gloeosporiums* and their relation to a Sweet-pea disease; Phytopath., II. BANGSOTT: A disease of Peppers; Jn. Bd. Ag. Brit. Guiana, VII. **Pepper. Stump Rot.**—SYDOW

& BUTLER: Fungi Indis Orientalis, III; Ann. Myc., IX, p. 404. NOWELL: Diseases of Lime trees in Forest districts; West Ind. Dept. Ag., Pamphlet Ser., No. 79. 12. Rosellinia root diseases in the Lesser Antilles; West Ind. Bul., XVI. **Coriander.** Tumours—BEEFELD: Untersuchungen, IX. v. BUREN: Zur Cytologie von Protomyces; Mycol. Centralb., IV. **Fennel.** Mildew.—SALMON: On Oldiopsis taurica (Lév.); Ann. Bot., XX.

## CHAPTER X.

## FIBRES.

**Cotton.** General.—ATKINSON: Diseases of Cotton: The Cotton Plant; U. S. A. Dept. Ag., Office of Expt. Stat., Bul. 33. EARLE: Diseases of Cotton; Alabama Expt. Stat. Bul. 107. HIBBARD: Cotton diseases in Mississippi; Miss. Expt. Stat. Bul. 140. LEWTON-BRAIN: Fungoid diseases of Cotton; West Ind. Bul., VI. v. FABER: Krankheiten der Baumwolle; Tropenpflanz., XII. Is: Die Krankheiten und Parasiten der Baumwollpflanze; Bakt. Centralb., 2nd Ab., XXIV. **Rust.**—ARTHUR: Uredinales; North Amer. Flora, VII, 3, p. 187. PETCH: Ustilaginæ and Uredinæ of Ceylon; Ann. Peraden., V, p. 247. **Anthracnose.**—EDGEINGTON: The Rots of the Cotton Boll; Louisiana Expt. Stat. Bul. 137. SMALL: Ann. Rep. of the Govt. Botanist in Ann. Rep. Dept. Ag., Uganda, 1917. Dept. of Ag., Burma, Cultivator's Leaflet 33, 1912. **Grey Mildew.**—POLK EVANS: Rep. of the Plant Pathologist in Ann. Rep. Transv. Dept. Ag., 1907-8, p. 5. SMALL: Ann. Rep. of the Govt. Botanist in Ann. Rep. Dept. Ag., Uganda, 1915. **Leaf Spot.**—MIYAKE: Ueber chinesische Pilze; Bot. Mag., Tokyo, XXVIII, p. 40. BALLS, in FODDEN & FLETCHER: Text Book of Egyptian Agriculture, II, p. 682. **Root Rot.**—SHAW: The morphology and parasitism of Rhizoctonia; Mem. Ag. Ind., Bot., IV, 6. PELTIER: Parasitic Rhizoctonias in America; Ill. Expt. Stat. Bul. 189. Sc. Rep. Ag. Res. Inst., Pusa, 1916-17, p. 60. **Jute.** **Root Rot.**—SHAW: l.c. **Sann Hemp.** **Rust.**—SYDOW & BUTLER: Fungi Indis Orientalis, II; Ann. Myc., V, p. 491. **Sisal.** **Anthracnose.**—SHAW: Anthracnose of Sisal Hemp; Ag. Jn. Ind., VIII. MORSTATT: Beobachtungen ueber des Auftreten von Pflanzenkrankheiten im Jahre 1911; Der Pflanze, VIII, 1912.

## CHAPTER XI.

## SUGARCANE.

General.—WAKKER & WENT: De Ziekten van het Suikerriet op Java. KRUEGER: Das Zuckerrohr und seine Kultur. COFFS: Fungus maladies of the Sugarcane; Expt. Stat. Hawaii Sugar Planters' Ass., Dn. Path. and Phys., Bula. 5 and 6. BUTLER: Fungus diseases of Sugarcane in Bengal; Mem. Ag. Ind., Bot., I, 3. DELACROIX & MAUBLANC: Les maladies des Plantes cultivées dans les Pays Chauds. (The chief references will be found in the above. The following are mostly additions.) SMUT.—AJREKAR: The mode of infection and prevention of the Smut disease of Sugarcane; Ag. Jn. Ind., XI. **Rust.**—BUTLER: Notes on some Rusts in India; Ann. Myc., XII. **Ring Spot.**—ASHBY: Diseases of Cocoas and other crops; Bul. Dept. Ag., Jamaica, II, 6. **Black Rot.**—PETCH: The Stem Bleeding disease of the Coconut; Circ. R. Bot. Gard., Ceylon, IV, No. 22, p. 247. PATTERSON, CHARLES, & VERMISTEY: Some Fungous diseases of economic importance; U. S. A. Dept. Ag., Bur. Pl. Ind., Bul. 171. **Dry Rot.**—PETCH: On Lasiodiplodia; Ann. Peraden., IV. BANUHOFF: The Me-back Fungus of Para Rubber and of Cacao (Thyridaria tarda n. sp.); Dept. Ag. F. M. S., Bul. '9. SYDOW & BUTLER: Fungi Indis Orientalis, V; Ann. Myc., XIV, p. 206. **Collar Rot.**—BUTLER & HAWES: Some new Sugarcane diseases; Mem. Ag. Ind., Bot., VI, 6. **Red Rot.**—BUTLER &

**HAFIZ:** Red Rot of Sugarcane; *Mem. Ag. Ind., Bot.*, VI, 5. *Will.*—**BUTLER & HAFIZ:** Some new Sugarcane diseases; *Mem. Ag. Ind., Bot.*, VI, 6. **NOWELL:** A stem disease of Sugarcane in Barbados; *Ag. News*, XV, pp. 14, 46. *Brown Leaf Spot.*—**KONUS:** Overzicht van het verloop der importatieplannen van vreemde Rietsorten op een eiland buiten Java; *Archief Java-Sulkerind.*, II, p. 661. *Helminthosporiose.*—**BUTLER & HAFIZ:** Some new Sugarcane diseases; *Mem. Ag. Ind., Bot.*, VI, 6. *Banded Sclerotial disease.*—**SAWADA:** Hypochnus on some cultivated plants in Formosa; *Bot. Mag., Tokyo*, XXVI.

## CHAPTER XII.

## TEA.

*General.*—**WATT & MANN:** The Pests and Blights of the Tea Plant, 2nd Ed., 1903. **DELAEROIX & MAUBLANC:** Maladies du Théier; *L'Agric. Prat. Pays Chauds*, VIII. **NIESCHNEW:** Die Pilzparasiten des Teestrauches, 1907. **BERNARD:** Notes de pathologie végétale; *Bul. Dept. Ag. Indes Néerl.* VI and XI. *Is.*: Observations sur le Thé, I, II; *ib.* XXIII. **TUNSTALL:** Notes; in *Quart. Jn. Ind. Tea Ass.* *Is.*: Tea Root, I, II; *Ind. Tea Ass. Bul.* I, 1916 and I, 1918. **ANDREWS & TUNSTALL:** Notes on the Spraying of Tea; *Ind. Tea Ass. Bul.* I, 1915. (The chief references will be found in the above. The following are mostly additions). *Red Rust.*—**MANN & HUTCHINSON:** *Cephaluros virescens* Kunze: the Red Rust of Tea; *Mem. Ag. Ind., Bot.*, I, 6. **BERNARD:** Eene ziekte van de Thee Plant veroorzaakt door *Cephaluros virescens*; *Meded. Proefstat. Thee, Buitenz.*, XXXII. **THOMAS:** Notes on *Cephaluros*; *Ann. Bot.*, XXVII. **VERMOESER:** Rapport sur quelques maladies cryptogamiques du Caocoyer au Mayumbe; *Bul. Ag. Congo Belge*, V. **ASHBY:** Diseases of Cocoes and other crops; *Bul. Dept. Ag. Jamaica, N. S.*, II, 6. *Blister Blight.*—**MORAE:** Report on the outbreak of Blister-blight on Tea in the Darjeeling District in 1908-9; *Bul. Ag. Res. Inst., Pusa*, XVIII, 1910. **SYDOW & BUTLER:** *Fungi Indis Orientalis*, IV; *Ann. Myc.*, X, p. 274-5. *Brown Root disease.*—(See under *Rubber Brown Root disease*). **PETCH:** Brown Root disease; *Circ. R. Bot. Gard., Ceylon*, V, 6. *Red Root disease.*—**PETCH:** A Root disease of Hevea; *Circ. R. Bot. Gard., Ceylon*, V, 8. **BROOKS:** Observations on some diseases of plantation Rubber in Malaya; *Ann. Appl. Biol.*, II. *Stump Rot.*—(See under *Coffee Stump Rot*). **PETCH:** Root diseases of Tea; *Trop. Agric.*, May, 1907, and *Circ. R. Bot. Gard., Ceylon*, V, 11. *Is.*: Revisions of Ceylon Fungi, II, p. 434, III, p. 286, IV, p. 22; *Ann. Peraden.*, IV-VI. **SYDOW & BUTLER:** *Fungi Indis Orientalis*, III; *Ann. Myc.*, IX, p. 405. **SHARPLES:** *Ustilina zonata*—A Fungus affecting Hevea brasiliensis; *Dept. Ag. F. M. S.*, *Bul.* XXV. v. **HÖRNEL:** Fragmente zur Mykologie, VI; *Sitz. K. Akad. Wiss., Wien, Math. Natur. Kl.*, CXVIII, 1, p. 343. **NOWELL:** Diseases of Lime Trees in Forest districts; *West Ind. Dept. Ag.*, Pamph. Ser., No. 79. *Is.*: Rosellinia root diseases in the Lesser Antilles; *West Ind. Bul.*, XVI. *Copper Blight.*—**SHAW:** The Copper Blight of Tea; *Ag. Jn. Ind.*, VI. *Internal Root disease.*—(See *Sugarcane Dry Rot and Rubber Die Back*). **SYDOW & BUTLER:** *Fungi Indis Orientalis*, V; *Ann. Myc.*, XIV, p. 205. **SOUTH:** Fungus diseases of Cacao; *West Ind. Bul.*, XII, p. 289. *Brown Blight.*—**SHEAR & WOOD:** Studies of Fungous parasites belonging to the genus *Glomerella*; *U. S. A. Dept. Ag., Bur. Pl. Ind.*, *Bul.* 252. **MORAE & ANSTAD:** Brown Blight of Tea; *Planters' Chron.*, XI, pp. 2, 43, 639. **SIMPSON:** *Ann. Rep. of the Govt. Botanist in Ann. Rep. Dept. Ag., Uganda*, 1916. *Grey Blight.*—**SAWADA:** Fungus diseases of the Tea Plant in Formosa (*Jap.*) *Spec. Rep. Ag. Expt. Stat.*, Formosa, II. **TANAKA:** New Japanese Fungi; *Myologia*, IX. **KLEBAHN:** Beiträge zur Kenntnis der Fungi imperfecti, III; *Mycol. Centralb.*, IV. **PETCH:** Report of the Govt. Mycologist; *Circ. R. Bot. Gard., Ceylon*, III, 21. **COLLETT:** Notes of observations of some Tea diseases; *Trop. Agric.*, XIX. **SIMPSON:** *Ann. Rep. of the Govt. Botanist in Ann. Rep. Dept. Ag., Uganda*, 1916.

*Leaf Spot*.—PETCH: Report of the Govt. Mycologist; Circ. R. Bot. Gard., Ceylon, III, 21. *Thread Blight*.—Rep. R. Bot. Gard., Ceylon, 1906 (Trop. Agric., XXIX, Suppl.). PETCH: Revisions of Ceylon Fungi, III; Ann. Peraden., V. *Is.*: Horse-hair Blights; *ib.*, VI. RICHARDS: Mycologist's Rep. for 1912-13; The Malay Penin. Ag. Ass. SAWADA: Hypochmus on some cultivated Plants in Formosa; Bot. Mag., Tokyo, XXVI. *Sooty Mould*.—SYDOW & BUTLER: Fungi Indis Orientalis, III; Ann. Myc., IX, p. 386. *Black Blight*.—SYDOW & BUTLER; *ib.*, p. 389. THE ISSER: Die Gattung Asterina; Abh. K. K. Zool. Bot. Gesells., Wien, VII, 3. *Canker*.—TUNSTALL: Fungus blights of Tea in north-east India during the season 1916; Quart. Jn. Ind. Tea Ass., 1917. *Seedling disease*.—HOPE & CARPENTER: Report on disease in Tea seed nurseries; Ind. Tea Ass., Bul. 6, 1909. PETCH: Miscellanea; Trop. Agric., XXXV, 3. BERNARD: Sur une maladie des jeunes Plantos de Thé; Bul. Dept. Ag. Indes Néerl., XL. *Seed Mould*.—ANTRAM: The Tea seed Bug; Quart. Jn. Ind. Tea Ass., I, 4; II, 1.

## CHAPTER XIII.

## COFFEE.

*General*.—DE LACROIX: Maladies des Cafésiers; L'Agric. Prat. Pays Chauds, VII—VIII. BUIR: Notes sur les maladies des Cafésiers; *ib.* VI. v. FABER: Die Krankheiten und Schädlinge des Kaffees; Bakt. Centralb., 2nd Ab., XXI—XXIII. ZIMMERMANN: Die Parasiten des Kaffees; *ib.*, V. *Is.*: Eenige pathologische en physiologische waarnemingen over Koffie; Meded. 's Lands Plantent., LXVII. FAWCETT: Fungus diseases of Coffee in Porto Rico; P. R. Expt. Stat. Bul. XVII. ANSTEAD, COLEMAN, and others: Numerous notes in Planters' Chronicle, esp. X—XIII. (The chief references will be found in the above. The following are mostly additions). *Leaf disease*.—FAUCHÈRE: Culture pratique du Caféier, &c.; L'Agric. Prat. Pays Chauds, VI—VII. v. FABER: Zur Infektion und Keimung der Uredosporen von Hemileia vastatrix; Ber. Deut. Bot. Gesells., XXVIII. *Is.*: Einiges ueber die Hemileia-Krankheit der Kaffeepflanze, &c.; Tropenpflanz., XIII. DE WILDEMAN: Les maladies du Caféier au Congo indépendant. C. R., 142. Coffee diseases in East Africa; Kew Bul., 1913, p. 168. The Coffee industry in the Island of Luzon; Phil. Ag. and For., I, 8. BARRETT: Coffee Blight in the Philippine Islands; Phil. Ag. Rev., V. POLE EVANS: Rep. of the Plant Pathologist in Ann. Rep. Transvaal Dept. Ag., 1906-7, p. 165. Informations diverses; Jn. d'Agric. Trop., XII, pp. 187, 220, 379. HARMAN: Report on Coffee Leaf disease (Hemileia vastatrix); Mysore, 1880. SYDOW & BUTLER: Fungi Indis Orientalis, I; Ann. Myc., IV, p. 440. CYRÉALIER: Le "Coffea excelsa" et sa culture; Jn. d'Agric. Trop., XIV, p. 193. FAUCHÈRE: Le Coffea congensia var. Chalotii à Madagascar; *ib.*, X, p. 1. CRAMER: L'influence de l'Hemileia vastatrix sur la culture du Café à Java; L'Agron. Trop., II. DE WILDEMAN: Matériaux pour une étude botanico-économique du genre Coffea; Ann. Jard. Bot. Buitenz., 3rd Suppl., 1910. VAN HALL: Robusta and some allied Coffee species; Ag. Bul. F. M. S., I. *Is.*: Gegevens over Robusta—en aanverwante Koffie-soorten; Teyssmannia, XXXIII. DOWSON: Ann. Rep. of the Mycologist in Ann. Rep. Dept. Ag., Br. E. Afr., 1913-16. SMALL: Ann. Rep. of the Govt. Botanist in Ann. Rep. Dept. Ag., Uganda, 1915-17. *Koleroga*.—v. HÖHNEL: Fragmente zur Mykologie, X; Sitz. K. Akad. Wiss., Wien, Math.-Natur. Kl., CXIX, 1, p. 395. COLEMAN: Black Rot of Coffee; Planters' Chron., X, 15. FAWCETT: Pellicularia Koleroga on Coffee in Porto Rico; Jn. Ag. Res., VI. KUYPER: Zilverdraadziekte der Koffie in Suriname; Dept. Landb. Sur., Bul. XXVIII. CAMERON: Report on Coffee Leaf disease in Coorg; Trop. Agric., XVIII. DE WILDEMAN: Les maladies du Caféier au Congo indépendant. C. R., 142. *Stump Rot*.—(See under *Tea Stump Rot*). PETCH: Root diseases of Tea; Circ. R. Bot. Gard., Ceylon V, II, p. 107. ANSTEAD: Root diseases; Planters' Chron., X, p. 156, XIII, p. 91. SMALL: Ann. Rep. of the Govt. Botanist in Ann. Rep. Dept. Ag., Uganda, 1915. *Leaf Spot*.—ASHBY: Diseases of Cocos and other crops; Bpl.



Dept. Ag., Jamaica, N. S., II, 6. KUYPER: Overzicht van de Koffieziekten in Suriname; Dept. Landb. Sur., Bul. XXXI. SIMPSON: Ann. Rep. of the Govt. Botanist in Ann. Rep. Dept. Ag., Uganda, 1916. *Brown Blight*.—SHEAR & WOOD: Studies of Fungous parasites belonging to the genus *Glomerella*; U. S. A. Dept. Ag., Bur. Pl. Ind., Bul. 252. MORSTATT: Die Schädlinge und Krankheiten des Kaffeebaumes in Ostafrika; Der Pflanze, Beiheft, VIII, 2. SIMPSON: l. c. *Brown Eye Spot*.—KUYPER: l. c. SIMPSON: l. c. *Sooty Mould*.—SYDOW & BUTLER: Fungi Indiæ Orientalis, III; Ann. Myc., IX, p. 384. SMALL: Ann. Rep. of the Govt. Botanist in Ann. Rep. Dept. Ag., Uganda, 1915.

## CHAPTER XIV.

## RUBBER.

*General*.—PETCH: The physiology and diseases of *Hevea brasiliensis*. *IB.*: The Fungus diseases of *Hevea brasiliensis*; Rubber Recueil. RUTGERS & ARENS: Diseases of *Hevea brasiliensis* in Java; *ib.* RUTGERS: Ziekten en Beschadigingen van *Hevea brasiliensis* op Java; Meded. Labor. Plantenziek., Buitenz., X. *Canker*.—RORER: Pod rot, Canker, and Chupon-wilt of Cacao caused by *Phytophthora* sp., Bul. Dept. Ag., Trinidad, IX, No. 65. PETCH: Cacao and *Hevea* Canker; Circ. R. Bot. Gard., Ceylon, V, 13. RUTGERS: *Hevea*-Canker, I—III; Meded. Labor. Plantenziek., Buitenz., II, IV, & XXVIII. GEBMANN: Krankheiten und Schädlinge der Kulturpflanzen auf Samoa; Arb. K. Biol. Anst., XI, 1. *Black Thread*.—McRAE: Note on *Phytophthora* Meadii on *Hevea brasiliensis*; Jn. Bom Nat. Hist. Soc., XXV. McRAE & ANSTEAD: Abnormal Leaf fall of *Hevea* Rubber; Planters' Chron., XIII, p. 38. DASTUR: *Phytophthora* sp. on *Hevea brasiliensis*; Mem. Ag. Ind., Bot., VIII, 5. RUTGERS: *Hevea*-Canker, I—III; Meded. Labor. Plantenziek., Buitenz., II, IV, & XXVIII. BELGRAVE: Notes on bark Cankers and their treatment; Ag. Bul. F. M. S., VI, 1. *IB.*: Further notes on Black-stripe Canker; *ib.*, VI, 3. RICHARDS: Diseases of the leaves and stem of *Hevea brasiliensis* in the Malay Peninsula; *ib.*, V. PRATT: Preventive measures against Black-thread; *ib.*, V. BRYCE: Diseases of *Hevea brasiliensis*; Trop. Agric., XLVIII. *Pink disease*.—BROOKS & SHARPLES: Pink disease; Dept. Ag., F. M. S., Bul. XXI. *IB.*: Pink disease of plantation Rubber; Ann. Appl. Biol., II. RORER: The Pink disease of Cacao; Bul. Dept. Ag., Trinidad and Tobago. XV. PETCH: Miscellaneous, Trop. Agric., XXXIII, 5. ZIMMERMANN: Ueber einige auf tropischen Kulturpflanzen beobachtete Pilze, I; Bakt. Centralb., 2nd Ab., VII. *Brown Root disease*.—(See under *Coffee Stump Rot*). BANCROFT: Brown root disease of Para Rubber; Ag. Bul., S. S. and F. M. S., April, 1911. BROOKS: Observations on some diseases of plantation Rubber in Malaya; Ann. Appl. Biol., II. *Die Back*.—(See *Dry Rot of Sugarcane and Internal Root disease of Tea*). PETCH: Die Back of *Hevea brasiliensis*; Circ. R. Bot. Gard., Ceylon, IV, 23. *IB.*: Descriptions of New Ceylon Fungi; Ann. Peraden., III. BROOKS: l. c. *Anthrax nose*.—KOORDERS: Botanische Untersuchungen ueber einige in Java vorkommende Pilze, &c.; Verh. K. Akad. Wet., Amsterdam, 2nd Sect., XIII, 4. SHEAR & WOOD: Studies of Fungous parasites belonging to the genus *Glomerella*; U. S. A. Dept. Ag., Bur. Pl. Ind., Bul. 252. *Hevea and Ceara Leaf Spots*.—PETCH: Descriptions of new Ceylon Fungi; Ann. Peraden., III. BANCROFT: A note on the leaf diseases of Para Rubber; Dept. Ag., F. M. S., Bul. XIV.



## INDEX.

- A**bsorption of food by fungi, 47, 50.
- Acacia**, *Corticium salmonicolor* on, 499 ; edible rust of, 91 ; woody gall of, 85.
- Acanthostigma parasiticum**, absorption of food by, 48.
- Acclimatisation**, influence on disease, 42, 117.
- Acervuli**, 18.
- Acids**, influence on parasites, 112, 121, 123, 503.
- Acropetal** formation of spores, 23.
- Æcidiospores**, 24, 29.
- Æcidium**, formation of, 29.
- Æcidium osculentum**, effect on inflorescence, 91 ; *Æ. Jacobsthalii*, effect on leaves, 90 ; *Æ. montanum*, 86 ; *Æ.* on coffee, 471.
- Agaricus melleus**, air requirements of, 112 ; resin flux produced by, 102 ; rhizomorphs of, 11.
- Agave** (*see* Sisal).
- Age of tissues** influences infection, 44, 68, 72, 119, 124, 472.
- Air**, influence on parasites, 97, 111.
- Alder trees**, *Valsa oxystoma* on, 117.
- Alkalies**, influence on parasites, 43, 112.
- Alternaria**, effect of nutrition on, 53 ; *A. Brassicæ*, 57, 81, 273, 300 ; *A. fasciculata*, 290 ; *A.* on cotton, 43 ; *A.* on melon, 300 ; *A. Solani*, 257, 304, 306.
- Amaranth** (*see* Chua).
- Amaranthus paniculatus** (*see* Chua).
- Amphicarpææ**, deformed nuclei of, 93.
- Anæsthetics**, influence on cereal mildew, 63 ; influence on cereal rusts, 125 ; (*see* Ether).
- Anastomoses**, 9.
- Anatomical factors** influencing disease, 72, 119.
- Anchor cells** of cobweb disease of coffee, 486 ; of thread blight of tea, 459.
- Andropogon halepensis**, diseases of, 206, 211, 212, 215, 218.
- Andropogon Sorghum** (*see* Jowar).
- Anemone rust**, dormant mycelium of, 58.
- Annual rings**, action of fungi on, 97, 112.
- Anthridium**, 25.
- Anthocyan** formed by fungal action, 93.
- Apium graveolens** (*see* Celery).
- Appendages** of perithecia of *Erysipheæ*, 174.
- Apple**, *Corticium salmonicolor* on, 499 ; mildew, 58 ; scab, 70, 72, 81, 100 (*see* *Venturia inæqualis*).
- Approssoria**, 68, 69.
- Arabis**, *Cystopus* on, 297.
- Arachis hypogæa** (*see* Groundnut).
- Areca palm**, *Fomes lucidus* on, 102.
- Argemone**, *Peronospora* on, 345.
- Arhar** (*see* Pigeon pea).
- Arrowroot**, sclerotial disease of, 411 ; *Sphaerostilbe repens* on, 435.
- Arthrobotryum** sp., sporophore and spores of, 17.
- Artocarpus**, *Corticium salmonicolor* on, 499.
- Asci**, formation of, 22, 25.
- Ascochyta** *Pisi*, 17, 205.
- Ascomycetes**, characters of, 36.
- Aeciospores**, 22, 24.
- Ancus**, 21.
- Aparagus rust**, influence of shade on, 111 ; resistance to, 126 ; specialisation of parasitism in, 62.
- Aspergillus niger**, mutants of, 53.
- Asterina Camellæ**, 462, 463.
- Asterocystis radialis**, localised infection by, 68.
- Atrophy**, 91.
- Avena fatua**, smut of, 182.
- Avena sativa** (*see* Oat).
- B**acillus coli on potato, action of, 43 ; *B. solanacearum*, 274, 303, 305, 333 ; gum produced by action of, 101, 102 ; *B. tumefaciens*, leafy shoots caused by, 89.
- Bacteria**, 4.
- Bagrada picta**, connection with coffee seed mould, 466.
- Bajra downy mildew**, 89, 218.
- rust, 223.

- Bajra smut, 224.  
 Bamboo, thread blight on, 457.  
 Barberry, rust of, 59, 60, 62, 154; witches' broom on, 86.  
 Barley covered smut, 184.  
     late blight, 187, 189.  
     loose smut, 184, 185.  
     mildew, 62, 120, 123, 124.  
     rust, 61, 63, 72, 119, 125, 185.  
     stripe disease, 166.  
 Basidiomycetes, characters of, 37.  
 Basidiospores, 24.  
 Basidium, 24.  
 Basipetal formation of spores, 23.  
 Bathua downy mildew, 317.  
 Bean anthracnose, 256; resistance to, 126.  
     leaf spot, 261.  
     powdery mildew, 256.  
     Rhizoctonia on, 263.  
     rust, 29, 290.  
 Beet leaf spot, 72.  
 Berberis, effect of *Æcidium Jacobsthalii* on, 90; (see Barberry).  
 Betel pepper (see Pepper).  
 Bhindi mildew, 306.  
 Biological species, 61.  
 Bordeaux mixture, 173.  
 Botryodiplodia Theobromæ, 383, 385, 445, 509.  
 Botrytis, effect of cuticle on infection by, 72; *B. cinerea*, action on cell walls, 96, 102; enzymic activity of, 68; influence of nitrate, 122; of reaction, 112; penetration assisted by organic debris, 66; toxic substances in, 51.  
 Brachypodium, cereal rusts on, 160.  
 Brachysporium stage of sooty moulds, 30.  
 Brassicas, blight, 81, 300.  
     downy mildew, 80, 297.  
     powdery mildew, 300.  
     white rust, 291.  
 Breadfruit, *Hymenochaete noxia* on, 430; *Phytophthora Faberi* on, 490; stump rot of, 359.  
 Bridging species, 63, 113.  
 Brinjal fruit rots, 305.  
     mildew, 271.  
     *Phytophthora Faberi* on, 490.  
     *Phytophthora parasitica* on, 330.  
     *Rhizoctonia* on, 263, 305.  
 Broccoli, *Peronospora parasitica* on, 91.  
 Brown oak, 52.  
 Buchloe, effect of *Tilletia* on, 88.  
 Buckle joint, 179.  
 Bud rot of palms, 76, 108.  
 Bulgaria polymorpha, action on cell walls, 97.  
 Burgundy mixture, 142.  
 Burrs on Hevea, 492.  
 Cabbage, *Fusarium*-resistant varieties of, 126; wilt, soil temperature and, 115; yellows, rotation for, 107; (see Brassicas).  
 Cacao, *Botryodiplodia Theobromæ* on, 386, 387, 447; brown root disease, 430; canker, 490; *Cephaluros Mycoidea* on, 414; *Corticium salmonicolor* on, 499; stump rot of, 359.  
 Cæoma, 324; *C. deformans*, galls of, 89; *C. Makinoi*, phylloidy in, 90.  
 Cajanus indious (see Pigeon pea).  
 Calcium oxalate, effects of fungus attack on formation of, 98.  
 Calluna, mycorrhiza of, 54.  
 Camellia Thea (see Tea).  
 Camphor, *Botryodiplodia Theobromæ* on, 386; *Corticium salmonicolor* on, 499; *Hymenochaete noxia* on, 430; *Rosellinia* on, 436, 438.  
 Canker, 82, 463, 490, 495, 501; treatment by excision of, 108, 493, 494.  
 Capnodium brasiliense, 30, 487; *C. Footii*, 462; *C.* on sugarcane, 411.  
 Capsella, *Cystopus* on, 93, 102; *Peronospora* on, 299.  
 Capsicum annum (see Chili).  
 Caraway, *Protomyces* on, 359.  
 Carica, *Glomerella piperata* on, 356; nuclei of *Vermicularia* on, 9; *Pythium gracile* on, 352.  
 Carnation rust, alteration in virulence of, 113.  
 Carotin in *Corticium salmonicolor*, 503.  
 Carrot, *Alternaria* on, 300; *Cercospora* on, 315; *Protomyces* on, 359.  
 Cassava leaf spot, 310.  
 Castilleja (see Rubber).  
 Castor leaf spot, 331.  
     *Phytophthora Meadii* on, 494.  
     *Pythium gracile* on, 352.  
     rust, 330.  
     seedling blight, 326.  
 Casuarina, *Trichosporium* on, 96.  
 Catenularia echinata, 383.  
 Cauliflower (see Brassicas).  
 Ceara rubber (see Rubber).  
 Celery, effect of *Phoma apiicola* on cells of, 94.  
 Celery-searly blight, 315.  
     leaf spot, 315; influence of shade on, 111.

- Cell contents, effects of fungus attack on, 93.  
 Cell wall, action of fungi on, 95.  
 Cellulase in fungi, 95.  
 Cellulose, 6; as food for fungi, 51, 95; defensive deposition of, 94, 281; dissolved by fungi, 51, 95.  
 Coltis, *Sphaerotheca* on, 68.  
 Cephalandra (see *Cucurbit*).  
 Cephaleuros Mycoidea, 413; on mango, 88.  
 Cephalosporium Sacchari, 402; anastomoses in, 9; sporiferous mycelium of, 17.  
 Cephalosporium stage of *Fusarium*, 247.  
 Coratostomella pilifera, insects that aid infection by, 35.  
 Croospora Apii, 315; influence of shade on, 111; *C. beticola*, influence of age on, 72; *C. canescens*, 261; *C. Ceras*, 516; *C. coffeicola*, 485; *C. conoidea*, 286; *C. cruenta*, 261; *C. Doliohi*, 261; *C. gossypina*, 369; *C. longipes*, 404; sporophores and spores of, 17; *C. Nicotianæ*, 340; *C.* on groundnut, 323; *C. personata*, 319; mycelium and haustoria of, 48; *C. Sacchari*, 406; *C. Theæ*, 455; *C. vaginæ*, appressoria of, 70; *C. Vignæ*, 261.  
 Crocosporina ricinella, 331.  
 Chemotropism, 64.  
 Chena rust, 238  
     smut, 235.  
 Chenopodium album (see *Bathua*).  
 Cherry, *Gnomonia erythrostoma* on, 104.  
 Chestnut bark disease, 34.  
 Chilli anthracnose, 356;  
     *Bacillus solanacearum* on, 338  
     die back, 352; influence of shade on, 111, 354.  
     mosaic disease, 342, 352.  
     ripe rot, 355.  
 Chitin in cell walls, 6.  
 Chlamydospores, 14.  
 Chlorophyll, 4; effects of fungus attack on, 79, 93.  
 Chua white rust, 316.  
 Cleor arietinum (see *Gram*).  
 Cinchona, *Corticium salmonicolor* on, 499, 503.  
 Cinnamomum, *Cephaleuros Mycoidea* on, 414; *Hymenochaete noxia* on, 430.  
 Cinnamon, *Corticium salmonicolor* on, 499.  
 Citrullus (see *Cucurbit*).  
 Citrus, die back, 484; foot rot, 84; *Glomerella piprata* on, 356; scab, 81; *Ustilina zonata* on, 440.  
 Cladosporium, peach scab caused by, 81;  
     *C. Chodati*, 230; *C. epiphyllum*, 43;  
     *C. herbarum*, 177, 207.  
 Clamp-connections, 9; function of, 27.  
 Classification of fungi, 36.  
 Clasterosporium, peach scab caused by, 81.  
 Claviceps, dissemination by insects of, 35;  
     *C. purpurea*, consumption of starch by, 52; specialisation of parasitism in, 62; (see *Ergot*).  
 Cleome, *Cystopus* on, 291.  
 Clerodendron, *Cladosporium* on, 43.  
 Climate, influence on disease, 117, 473.  
 Clover, anthracnose-resistance in, 126;  
     *Sclerotinia Trifoliorum* on, 70.  
 Cluster-bean (see *Guar*).  
 Coagulase in fungi, 52.  
 Coca, *Corticium salmonicolor* on, 499;  
     *Hymenochaete noxia* on, 430.  
 Coccinia (see *Cucurbit*).  
 Coconut, bleeding disease, 84, 385;  
     *Botryodiplodia Theobromæ* on, 396;  
     *Pestalotzia palmarum* on, 451; *Ustilina zonata* on, 440.  
 Coffee species in relation to leaf disease, 473, 474.  
 Coffee anthracnose, 482.  
     *Botryodiplodia Theobromæ* on, 386.  
     brown blight, 482.  
     brown eye spot, 485.  
     brown root disease, 480.  
     *Cephaleuros Mycoidea* on, 414.  
     cobweb disease, 486.  
     die back, 483.  
     koleraga, 58, 477; influence of moisture on, 70.  
     leaf disease, 467, 468; alterations in virulence of, 113; conditions affecting severity of, 73, 473; influence of soil on, 117; of vigour of bush on, 76, 115, 470.  
     leaf spot, 481.  
     pink disease, 479.  
     root diseases, 479.  
     Rosellinia on, 436.  
     rust, 466.  
     seed mould, 466.  
     sooty mould, 30, 437.  
     stump rot, 359, 479.  
     twig disease, 84, 493.  
 Coix *Lachryma-Jobi* (see *Kadi*).  
 Colletotrichum Agaves, 374; *C. Camellia*, 443, 448, 455; *C. Carveri*, 446; *C. coffeanum*, 482; *C. falcatum*, 391; appressoria of, 69; infection by, 66, 67; inverted cane

- sugar, 51; *C. Ficus*, 513; *C. graminicolum*, 217; *C. incarnatum*, 485; *C. Lineola*, 217; *C. Lindemuthianum*, 256; *C. nigrum*, 356; *C. phomoides*, 304.
- Colocasia antiquorum* (see Kachu).
- Columella, 209.
- Conidia, 24.
- Coniferin, 96.
- Coniothecium stage of sooty moulds, 30.
- Coniothyrium, waxy varieties resist, 119.
- Copper carbonate spray fluid, 143.
- Copper spray fluids, risk of poisoning by, 137.
- Copper sulphate treatment for smuts, 169, 181, 211, 235, 237.
- Corchorus (see Jute).
- Coriander mildew, 361.
- tumours, 359.
- Cork, action of fungi on, 99, 100, 103, 104; formation of, 99; influence on penetration, 72; *Phytophthora infestans* may penetrate, 282.
- Corticium Koleroga, 477; absorption of food by, 47; *C. salmonicolor*, 429, 479, 499; influence of shade on, 72; thyloses caused by, 102; *C. Sasakii*, 410; *C. Solani*, 264; *C. Theae*, 460.
- Coryneum Mori, frost may aid infection by, 67.
- Cotton bolls, dropping of, 84.
- disease resistance in, 114, 126.
- Alternaria* on, 43.
- anthracnose, 365.
- grey mildew, 368.
- Hymenochaete noxia* on, 430.
- leaf spot, 369.
- mildew, 363.
- root rot, 370.
- rust, 363.
- wilt, 107, 126.
- Cowpea, *Fusarium*-resisting varieties of, 126.
- anthracnose, 262.
- die back, 262.
- leaf spot, 261.
- powdery mildew, 262.
- Rhizoctonia*, 12.
- root rot, 262.
- rust, 260, 262.
- Cress, *Cystopus* on, 293, 297.
- Crotalaria, *Corticium salmonicolor* on, 499.
- Crotalaria juncea* (see Sann hemp).
- Crown-gall, effect on tobacco leaves of, 89.
- Cucumber (see Cucurbit).
- Cucurbit downy mildew, 311.
- powdery mildew, 314; influence of shade on, 111.
- root rot, 315.
- Cultivation, influence on coffee leaf disease, 473; on tea red rust, 422.
- Curcuma longa* (see Turmeric).
- Curcuma* spp., *Taphrina* on, 347.
- Custard apple, *Corticium salmonicolor* on, 499.
- Cuticle, position of *Diplocarpon Rosae* below, 94, 99.
- Cutin, action of fungi on, 99, 102.
- Cutinase in fungi, 52.
- Cyamopsis psoraloides* (see Guar).
- Cyclogonium oleaginum, digests cutin, 99; lignification caused by, 100.
- Cystopus Bliti*, 316; *C. candidus*, 291; attacks only healthy plants, 45; influence of temperature on infection by, 71; nutrient tissue in plants attacked by, 294; specialisation of parasitism in, 61, 297; *C. Ipomoeae panduranae*, effect on host of, 90; *C. Portulacae*, 344.
- Cytase in fungi, 52, 95.
- Dahlia, *Botrytis cinerea* on, 66.
- Damping off, 79; of jute, 373.
- Datura*, *Alternaria Solani* on, 290; *Bacillus solanacearum* on, 338.
- Defensive reaction, scheme of, 100.
- Defoliation, 84.
- Delignification by fungi, 97.
- Delphinium, *Rhizoctonia destruens* on, 291.
- Deodar, rhizomorphs of *Fomes annosus* on, 11; witches' broom of, 87.
- Deterioration in disease resistance, 127.
- Deuteromycetes, characters of, 37.
- Diastase in fungi, 52.
- Die back, 79.
- Diplocarpon Rosae*, consumes cellulose, 95; outmination caused by, 100; defoliation by, 88; sub-cuticular growth of, 94, 99.
- Dissemination of fungi, 32.
- Division of cells provoked by fungi, 100, 104.
- Dolichos (see Val).
- Dolichos sesquipedalis*, leaf spot of, 261.
- Dormant state of mycelium, 45, 57, 58, 160.
- Doubling of flowers caused by fungi, 89.
- Drying of spores may aid germination, 71.
- Duan, disease of, 291.
- Metoparaasites, 46.
- Eleusine coracana (see Ragi).

- Embryonic tissue, parasites that attack, 44, 68.
- Emulsin in fungi, 52, 97.
- Endogenous formation of spores, 20.
- Endoparasites, 46.
- Endothia parasitica, tannin as food for, 52; (*see* Chestnut bark disease).
- Enzymatic virus of mosaic disease, 342.
- Enzymes in fungi, 51, 52, 95, 97.
- Enzymic activity, greatest in germ-tubes, 68.
- Epichloe cinerea, atrophy caused by, 91.
- Epidemics, 129.
- Eragrostis tenuifolia, Epichloe on, 91.
- Ergot, disseminated by insects, 35; infects through ovary, 68; specialised races of, 62; (*see* Claviceps).
- Eruca sativa (*see* Duan)
- Erysiphaceæ, absorption of food by, 49; dormant mycelium of, 58; influence of climate on perfect stage of, 31.
- Erysiphe Cichoracearum, 306, 314, 338; attacks only healthy plants, 45, 116; influence of light on, 111; *E. graminis* on wheat, 173; specialisation of parasitism in, 62, 175; *E. Polygoni*, 253, 256, 262, 300, 361, 362; haustoria of, 69; appressoria of, 69; specialisation of parasitism in, 61, 254; *E. taurica*, 271.
- Erythrina, Botryodiplodia Theobromæ on, 386; Corticium salmonicolor on, 499; Hymenochaete noxia on, 430; Rosellinia on, 438; Sphaerostilbe repens on, 435.
- Ether, influence on infection, 46, 63, 116, 124, 125.
- Eucalyptus, Corticium salmonicolor on, 499.
- Euchlena mexicana, diseases of, 193, 194, 199, 218, 223.
- Euonymus mildew, 33, 58, 125.
- Euphorbia, rust, dormant mycelium of, 58; Uromyces on, 91, 256.
- Eurya, Exobasidium on, 85.
- Exoascaceæ, parasitic life of, 49.
- Exoascus Cerasi, mucilaginous degeneration caused by, 102; *E. deformans*, alterations caused by, 92; mycelium of, 48, 49, 58; *E. Pruni*, hypertrophy due to, 86.
- Exobasidium Euryæ, hypertrophy due to, 85; *E. vexans*, 422; infection by, 65, 66.
- Exogenous formation of spores, 23.
- Exudations caused by fungi, 83, 102.
- Fat and oil in fungus cells, 8.
- Fats split by fungi, 51.
- Fennel mildew, 361.
- Fenugreek mildew, 362.
- rust, 362.
- Ferments in fungi, 51, 52, 95, 97.
- Fertilisers, influence on susceptibility to disease, 63, 122; (*see* Manure, Nitrogen, Phosphates, Potash).
- Festuca, Puocinia graminis on, 160.
- Ficus, Hymenochaete noxia on, 480; Rosellinia on, 436; (*see* Rubber).
- Finger and toe, rotation for, 107; (*see* Plasmodiophora).
- Flax, infection in brûlure of, 65; retting of, 96; rust of, 324; resistance of linseed to rust of, 126; wilt of, resistance to, 114, 126; rotation for, 107; (*see* Linseed).
- Florida bean (*see* Velvet bean).
- Flower infection, 35, 68.
- Fluxes, 83.
- Fœniculum vulgare (*see* Fennel).
- Fomes annosus, alterations in wood caused by, 96; exudation of resin caused by, 84; dissemination by animals, 35; infection by, 67; rhizomorphs of, 11; *F. carneus*, consumes cellulose, 95, 96; *F. juniperinus*, action on cell walls, 97; *F. lignosus*, 441, 506, 511; *F. lucidus*, thyloses caused by, 102; *F. nigricans*, action on cell walls, 97; wood gum not affected by, 95, 97; *F. punctata*, consumes cellulose, 96; *F. rimosus*, destroys medullary rays, 97; *F. semitostus*, 441, 506, 507; *F. texanus*, action on cell walls and wood, 97.
- Food of fungi, 41.
- Foot rot of orange, 78; treatment by excision, 108.
- Formalin treatment for smuts, 170, 181, 211, 235, 237.
- Freezing of spores may aid germination, 71.
- French bean (*see* Bean).
- Frost, cankers caused by, 82; defoliation caused by, 84; may aid infection, 63, 67, 118; may kill branches, 79.
- Fruit, dropping of, 84.
- Fruit rots, plasmolysis in, 93; prevention of, 109; weak parasites may cause, 83.
- Fungicides, 131.
- Fusarium, action of potato-rotting species of, 93, 95; *F. Hordearium*, consumes cellulose, 95; *F. Lycopersici*, 304; *F. udum*, 244; anastomoses in, 9; chlamydospores of, 14.
- Fusarium wilts, dissemination of, 35; plants resistant to, 125; relation to soil

- of, 112; rotation for, 117; saprophytic life of, 44.
- Fusicladium*, action on cell wall, 99; (*see* *Venturia*).
- Galactinia succosa*, nuclei of, 9.
- Gall-formation by fungi, 51.
- Galls, 84; infection of, 68, 124.
- Garlic mildew, 271.
- Gommæ, 14.
- Geranium*, *Venturia* on, 102.
- Germ-tubo, 6.
- Ginger soft rot, 348.
- Gliosporium alborubrum*, 510, 511; *G. coffeanum*, 482; *G. Elasticæ*, 512; *G. Lindemuthianum*, 256; *G. piperatum*, 355; *G. Theæ*, 451.
- Glomereella cingulata*, 262, 304, 355, 443, 448, 482, 512; pentosanase in, 95; *G. Gossypii*, 365; *G. Lindemuthianum*, 256, 262, 267; specialisation of parasitism in, 61, 260; *G. Lycopersici*, 304; *G. piperata*, 304, 306, 355.
- Glucosidos split by fungi, 51, 97.
- Glycine hispida* (*see* Soy bean).
- Glycogen in fungus cells, 8.
- Gnomonia erythrostoma*, action on leaves, 104.
- Gooseberry mildew, American, 34; influence of nitrogen on, 122.
- Gossypium* (*see* Cotton).
- Gourd (*see* Cucurbit).
- Grape (*see* Vine).
- Gram blight, 268.
- rust, 271.
- wilt, 107, 271.
- Grevillea*, *Corticium salmonicolor* on, 499; *Hymenochaete noxia* on, 430, 479; *Rosellinia arcuata* on, 436; *R. bunodes* on, 359; *Ustilina zonata* on, 438.
- Groundnut, *Bacillus solanacearum* on, 338.
- root rot, 323.
- tikka disease, 319.
- Groundnuts, deterioration in, 128.
- Guar leaf spot, 273.
- mildew, 271.
- Guignardia Bidwellii*, does not consume starch, 52; effect on cell contents, 93; hypertrophy caused by, 104; influence of weather on, 444; sub-cuticular growth of, 99; (*see* Vine black rot).
- Gum, exudation of, 64, 102.
- Gum-formation in tobacco bacterial wilt, 101, 335.
- Gummosis, 102.
- Gummy degeneration, 9, 101, 102.
- H**
- Hadromal*, 96.
- Hadromase* in fungi, 52, 97.
- Hematochrom* in *Cephaleuros*, 417.
- Hairs may prevent infection, 72.
- Hapalophragmium ponderosum*, gall of, 85.
- Haustoria*, 46, 48, 49, 50, 69.
- Hawthorn, *Roestelia* on, 104.
- Heart-wood, action of fungi on, 95, 97.
- Helleborus*, dormant mycelium in, 58.
- Helminthosporium Avenæ*, 183, 187; *H. gramineum*, 186; *H. Heveæ*, 515; *H. nodulosum*, 241; *H. Sacchari*, 406; germination of spore of, 7; *H. teres*, 187, 189; *H. turcium*, 201, 218.
- Hemi-parasites, 42.
- Hemi-saprophytes, 42.
- Homileia Canthii*, 468; *H. vastatrix*, 468; appressoria of, 69, 70, 472; influence of light on, 71, 472; (*see* Coffee leaf disease); *H. Woodii*, 468.
- Hendersonia theicola*, 455.
- Hendersonina Sacchari*, 398; anastomoses in, 9.
- Heterocism, 59.
- Hevea* (*see* Rubber).
- Hibernating mycelium, 58.
- Hibiscus esculentus* (*see* Bhindi).
- Holigarna, *Rosellinia bunodes* on, 358.
- Hollyhook rust, alteration in virulence of, 113; dormant mycelium of, 45, 58; mycoplasma of, 161.
- Honey dew, 30, 411, 487.
- Hordeum sylvaticum*, mildew of, 62, 124, 175.
- Hordeum vulgare* (*see* Barley).
- Hormodendron cladosporioides*, 178.
- Horse-hair fungi, 11.
- Host plant, 42.
- Hot-water treatment for smuts, 165, 181, 185, 211, 238.
- Humidity, influence on disease, 110, 117.
- Humin bodies produced by action of *Merulius*, 95.
- Hybridisation and disease, 126.
- Hybrids may act as bridging species, 64.
- Hydrotoplasma, 64.
- Hymenium, 19.
- Hymenochaete noxia*, 429, 430, 506.
- Hypoxyama*, *Alternaria Solani* on, 290.
- Hypertrophy, 84, 108.
- Hypha, 6.
- Hyphopodia, 462, 463.



- Hypochnus Saakii*, 410; *H. Solani*, 202, 290, 305, 315, 323; basidia and sporidia of, 21.
- Immunity**, 114
- Incubation period**, 57; may vary with age of tissues, 472; with temperature, 45, 160.
- Indigo**, *Bacillus solanacearum* on, 338; *Botryodiplodia Theobromae* on, 385; *Corticium salmonicolor* on, 499; damping off of, 79
- Infection**, 64.
- Inflorescence altered by fungus attack**, 91.
- Insects may aid infection**, 35, 63, 67, 116, 401, 454; may prevent infection, 45, may disseminate fungi, 35.
- Intumescences caused by *Bacillus solanacearum* in tomato**, 89.
- Invertase in fungi**, 52
- Ipomoea**, *Cytopus* on, 90.
- Iris**, *Heterosporium gracile* on, 124
- Italian millet (see Kangni)**
- Jasmine**, *Corticium salmonicolor* on, 499.
- Uromyces** on, 103
- Job's Tears millet (see Kasi)**
- Jowar downy mildew**, 203
- grain smut, 208.
- head smut, 214
- leaf blight, 218
- long smut, 215
- loose smut, 212
- red leaf spot, 217
- rust, 206.
- Jute**, damping off of, 79; rotting of, 96; *Rhizoctonia* of, 12, 372
- Jute**, root rot, 372
- Kachu blight**, 306
- Kangni green ear disease**, 232
- rust, 233.
- smut, 234.
- Kasi black leaf spot**, 243
- rust, 242
- smut, 242.
- Khesari downy mildew**, 268
- rust, 268.
- Kodra rust**, 240
- smut, 240.
- Kuehneola Fici**, promycelium and sporidia of, 21; *K. desmii*, 364.
- Kulfa white rust**, 318.
- Kulthi (see Val)**.
- Kutki rust**, 238.
- Laccase in fungi**, 52.
- Laetitia Bidwellii**, 444 (see *Guignardia*); *L. Camelliae*, 443; *L. Theae*, 443.
- Lagenaria (see Cucurbit)**.
- Lantana**, *Corticium salmonicolor* on, 499.
- Larch canker**, attacks young trees most, 119; influence of soil on, 117, 124; insects that aid infection by, 35.
- Latex**, exudation of, 84.
- Lathyrus sativus (see Khesari)**.
- Lathyrus sylvestris**, downy mildew of, 252.
- Launea asplenifolia**, rust of, 58, 90.
- Leaf fall**, 84, 494.
- Ledum**, *Sclerotinia* on, 59
- Legislation against fungus diseases**, 36
- Lens esculenta (see Lentil)**.
- Lentil**, *Rhizoctonia destruens* on, 291.
- rust, 29, 208
- Lentinus lepideus**, action on cell walls, 97.
- Lenzites sepiaria**, action on wood, 98
- Leptosphaeria herpotrichoides**, prevents thickening of walls, 102; *L. Sacchari*, 21, 387; *L. Tritici*, 175; infection by, 66
- Lettuce mildew**, etiolation predisposes to, 120; phosphates predispose to, 123
- Lichens**, 5, 54.
- Light**, influence on disease, 111; influence on spore germination, 71, 472.
- Lignification**, 96.
- Lignified walls**, action of fungi on, 96.
- Lilac**, *Polystictus vocifericolor* on, 97.
- Limacinaula Theae**, 461.
- Lime black root disease**, 359, 436.
- Lime**, influence on disease, 124; used for soil disinfection, 135.
- Lime-sulphur spray fluids**, 144.
- Linseed rust**, 324.
- Linum usitatissimum (see Flax, Linseed)**
- Lipase in fungi**, 52
- Litsea**, *Rosellinia bunodes* on, 358.
- Lolium temulentum**, symbiotic fungus of, 53.
- Loquat**, *Corticium salmonicolor* on, 499; thread blight on, 457.
- Luffa (see Cucurbit)**.
- Lychnis smut**, 58, 88.
- Lycopersicum esculentum (see Tomato)**.
- Macrosporium Lycopersici**, 306; *M. Tomato*, 290, 304, 306.
- Maize downy mildew**, 191.
- head smut, 199.
- leaf blight, 201.
- rust, 193.
- smut, 194; generalised infection by, 46, 116, 124.

- Mandua* (see Ragi).
- Mango**, *Botryodiplodia Theobromae* on, 386; *Corticium salmonicolor* on, 499; effect of *Cephaleuros Mycoidea* on, 88; red rust, 100, 414; thread blight, 457.
- Mangold rust**, 123.
- Manihot Glazovii** (see Rubber).
- Manihot piauhyensis**, 516.
- Manihot utilisima** (see Cassava).
- Manuro**, diseases carried by, 109, 169; influence on coffee leaf disease, 474; influence on jute root rot, 373; influence on potato blight, 284; influence on tobacco wilt, 337.
- Maranta**, sclerotial disease of, 411; *Sphaerostilbe repens* on, 435.
- Mareuola Juglandis**, acervulus of, 17.
- Matricaria**, *Peronospora* on, 90.
- Medicago**, downy mildew of, 266.
- Medullary rays**, action of fungi on, 97, 103.
- Melampsora Lini**, 324.
- Melampsorella Ricini**, 330.
- Melanopsichium austro-americanum**, pro-mycelium and sporidia of, 21.
- Melanospora damnosa**, influence of soil on, 124.
- Melilotus**, downy mildew of, 266.
- Melon**, *Alternaria* on, 300; (see Cucurbit, Watermelon).
- Mercurialis**, *Synchytrium* on, 104.
- Merulius lachrymans**, hadromase in, 97; humin formed by, 95.
- Micron**, 65.
- Middle lamella**, action of fungi on, 95, 119.
- Moisture**, influence on black thread of rubber, 498; on blister blight of tea, 429; on Brassica blight, 302; on coffee leaf disease, 473; on Colocasia blight, 308; on copper blight of tea, 444; on cucurbit mildew, 313; on infection, 70, 110, 119; on pink disease of rubber, 503; on potato blight, 283; on spore germination, 70; on tobacco mildew, 339; on tobacco wilt, 336.
- Momordica** (see Cucurbit).
- Monilia fructigena**, penetration assisted by fruit juices, 66.
- Mosaic disease**, 341.
- Moth** (see Bean).
- Mucor**, effect of nutrition on, 53; *M. Prainii*, gemmae of, 14; mycelium of, 8.
- Mucuna** (see Velvet bean).
- Mulberry**, *Coryneum Mori* on, 67; *Nectria cinnabarina* on, 67; *Polyporus hispidus* on, 108.
- Mung** (see Bean).
- Mustard** (see Brassicas).
- Mycelium**, 6.
- Mycoeloderma cuticularis**, cell division induced by, 101.
- Mycoplasma**, 161.
- Mycoorhiza**, 54.
- Mycosphaerella coffeicola**, 481; *M. Fragariae*, influence of shade on, 111; *M. gossypina*, 369; *M. pinodes*, 268; *M. Tulaneii*, 177.
- Mylitta lapidescens**, 13.
- Myxomycetes**, 5.
- Necator decretus**, 501.
- Nectria Bolbophylli**, 31, 249; perithecium of, 17; *N. cinnabarina*, on tea, 463; frost may aid infection by, 67; thylosem caused by, 102; *N. ditissima*, 463; *N. galligena*, may enter through lenticels, 67.
- Nectrias**, conveyed by insects, 35.
- Neocosmospora vasinfecta**, 32, 245, 249; asci and ascospores of, 21; conidia of, 21.
- Neozimmermannia Elasticæ**, 512.
- Nicotiana** (see Tobacco).
- Nitrogen**, influence on disease, 122, 177, 284.
- Nucleus**, 7, 9; effects of fungus attack on, 93, 94.
- Nutrition**, effects on fungi of different, 52; influence on disease, 122.
- Oak mildew**, alteration in virulence of, 113; effect of factory smoke on, 116; mycelium of, 48.
- Oat leaf spot**, 183, 187.
- mildew**, 62.
- rust**, 180, 182.
- smut**, 170, 180.
- Oidiopsis taurica**, 271, 362; *O. taurica* var. *lanuginosa*, 361.
- Qidium Balsami**, 300; *O.* on cotton, 363.
- Oogonium**, 25.
- Oospore**, potato scab caused by, 81.
- Oospore**, 25.
- Opium poppy blight**, 344.
- downy mildew**, 344.
- root rot**, 346.
- Orange blue mould**, 83; *Botryodiplodia Theobromae* on, 385; *Corticium salmonicolor* on, 499; foot rot, 78, 108.
- Orchids**, mycorrhiza of, 54.
- Oryza sativa** (see Rice).
- Palm**, bud rot of, 76, 108.
- Palmyra palm disease**, 34.

- Panicum Oriz-galli*, smut of, 238.  
*Panicum frumentaceum* (see *Sawar*).  
*Panicum miliaceum* (see *Chena*).  
*Panicum miliare* (see *Kutki*).  
*Panicum repens*, rust of, 238.  
*Panicum spinosens*, smut of, 238.  
*Papaver somniferum* (see *Opium*).  
*Papaver* spp., *Peronospora* on, 345.  
*Papaya*, *Botryodiplodia Theobromae* on, 386; *Phytophthora Faberi* on, 490; (see *Carica*).  
*Para* rubber (see *Rubber*).  
*Paraphyses*, 19.  
*Parasites*, 42; life-history of, 56.  
*Parasitism*, origin of, 43.  
*Parasip*, *Cercospora* on, 315; *Protomyces* on, 359.  
*Paupalum scrobiculatum* (see *Kodra*).  
*Pea* downy mildew, 251.  
     powdery mildew, 253.  
     rust, 29, 254.  
*Peach* leaf-curl, 48, 49, 58, 92; mildew, 45; scab, 81; shot hole, 81.  
*Pear*, *Fusarium putrefaciens* on, 119; scab, 70, 81, 100 (see *Venturia pirina*).  
*Pectic* bodies dissolved by fungi, 51, 95.  
*Pectinase*, 52, 97.  
*Polargonium*, *Botrytis cinerea* on, 66.  
*Pellicularia Koleroga*, 477.  
*Penetration* of plants by fungi, 64.  
*Penicillium*, effect of nutrition on, 53; germination of spore of, 6; in tea seed, 465; may attack fruit, 83; *P. glaucum*, penetration of living plants by, 65.  
*Pennisetum typhoideum* (see *Bajra*).  
*Pentosanase* in fungi, 95.  
*Pepper*, *Corticium salmonicolor* on, 499.  
     *Rhizoctonia destruens* on betel, 291.  
     stamp rot, 357.  
*Peptonising* action of fungi, 51.  
*Perennial mycelium*, 58, 86.  
*Perfect* stage of a fungus, 25; late development of, 50.  
*Peridermium*, resin flux produced by, 84, 102; *P. Cedri*, witches' broom caused by, 87.  
*Peridium*, 29.  
*Perithecium*, 19.  
*Peronospora*, preference for healthy plants, 45; *P. arborescens*, 344; *P. effusa*, 317; *P. farinosa*, 318; *P. parasitica*, 80, 297; infection by, 45, 66; leaf spotting by, 80; reversion caused by, 91; *P. Radii*, effect on flowers, 91; *P. Trifoliorum*, 266; *P. Trifoliorum* var. *manshurica*, 266; *P. Viciae*, 17, 251, 268.  
*Pestalotzia Guopini*, 413, 451; *P. palm-arum*, 413, 451; *P. Theae*, 451.  
*Phalaris*, *Puccinia glumarum* on, 180.  
*Phalloids* disseminated by flies, 35.  
*Phascolus* (see *Bean*).  
*Phoma apicola*, effect on cello, 94; *P. Camelliae*, 443.  
*Phosphates*, influence on disease, 112, 123, 177, 284.  
*Phototropism*, 64.  
*Phycomycetes*, characters of, 36.  
*Phyllachora Coioui*, 443.  
*Phyllody*, 90, 104.  
*Physiological* effects of spraying, 138.  
*Physiological* factors influencing disease, 73, 120.  
*Physiological* species, 60.  
*Phytophthora Colocassiae*, 306; chlamydospores of, 14; influence of light on, 71; haustoria of, 50; infection by zoospores of, 65; *P. erythrocephala*, 282, 286; *P. Faberi*, 490; *P. infestans*, 277, 304 (see *Potato blight*); *P. Meadii*, 494; *P. "omnivora"*, consumption of starch by, 52; *P. palmivora*, solution of cellulose by, 94, 95; *P. parasitica*, 326; influence of light on, 71.  
*Pigeon pea* wilt, 244.  
*Pine*, *Fraxinus annosus* on, 96; *Stereum hirsutum* on, 97; *Trametes Pini* on, 97; (see *Pinus*).  
*Pine-apple*, *Botryodiplodia Theobromae* on, 386; disease of sugarcane, 385; *Thielaviopsis paradoxa* on, 385.  
*Pinus*, *Ceratostomella* on, 35; *Trametes Pini* on, 83.  
*Piper nigrum* (see *Pepper*).  
*Pisum* (see *Poa*).  
*Plantain*, *Glomerella piperata* on, 356.  
*Plasmidiophora Brassicae*, effect on plant, 92, 103; lime treatment of, 135.  
*Plasmolysis*, 63.  
*Plasmopara viticola*, sub-stomatal vesicle of, 66.  
*Pleolpidium irregulare*, infection by, 65; *P. Pythii*, sporangia and zoospores of, 21.  
*Pleospora trichostoma*, 188.  
*Plum*, *Corticium salmonicolor* on, 499; *Sclerotinia cinerea* on, 119; shot hole of, 81.  
*Pseudocoris latus*, connection with tea seed mould, 465.  
*Polydesmus exitiosus*, 300.

- Polygonum*, *Ustilago* on, 89, 104.  
*Polymorphism*, 27.  
*Polyporus hispidus*, basidia and sporidia of, 21; treatment of, 108; *P. ignarius*, attacks starch, 93; *P. Mylittæ*, 13; *P. Schweinitzii*, consumes cellulose, 95; *P. Shoreæ*, action on sap-wood, 99; *P. squamosus*, action on wood, 97; enzymes of, 52; *P. subacidus*, action on wood, 97, 98; *P. sulphureus*, action on wood, 97; diastatic action, 51.  
*Polystictus pergamenus*, action on wood, 97; *P. sacer*, 13, 18; *P. versicolor*, action on wood, 97.  
 Poppy bacterial disease, 346.  
     downy mildew, 314.  
     root rot, 346.  
*Poria hypolateritia*, 435; *P. vaporaria*, consumes cellulose, 95.  
*Portulaca oleracea* (see Kulla).  
 Potash, influence on disease, 123, 284, 373.  
 Potassium sulphide treatment for smut, 181.  
 Potato, *Bacillus coli* on, 43.  
     bacterial wilt of, 274.  
     blight, 277; climatic conditions influencing, 71, 117, 283; factors that cause epidemics of, 129; influence of nitrogen on, 122, 284; rapidity of spread of, 106; resistance to, 126, 127.  
     *Botryodiplodia Theobromæ* on, 386.  
     early blight, 287.  
     leaf blotch, 286.  
     *Phytophthora Colocasiæ* on, 306.  
     *Phytophthora erythroseptica* on, 268.  
     *Phytophthora parasitica* on, 330.  
     ring disease, 74.  
     root rot, 290.  
     rotting, action of *Fusarium* in, 93, 95.  
     scab, 81.  
     tuber rot, 290.  
     wart disease, infection in, 65.  
 Potatoes, deterioration in, 127; disease resistance in, 114, 115, 119, 122, 125, 286; storing of, 109, 284.  
 Predisposition, 114.  
 Proliferation, 89.  
 Promycelia, asprophytism of, 60.  
 Promycelium, 24.  
 Protease in fungi, 52.  
 Protomyces, specialisation of parasitism in, 61; *P. macrosporus*, 359; tissue alterations caused by, 104.  
*Prunings*, danger from, 107.  
*Prunus*, *Craoma* on, 90; *Exosascus* on, 86.  
*Pseudococcis*, 93.  
*Pseudoparenchyma*, 10.  
*Pseudoperonospora cubensis*, 311.  
*Pteris*, *Taphrina* on, 89.  
*Puccinia Butleri*, effect on host of, 90; (see *Launca*); *P. Chrysanthemi*, growth on resistant hosts, 121; *P. coronata* may reduce calcium oxalate, 93; *P. cornifera*, 182; *P. dispersa*, appressoria (cf. 69; *P. glumarum*, 151, 185; specialisation of parasitism in, 186; *P. graminis*, 151, 182, 185; barberry not essential to, 60, 159; growth on resistant hosts, 63, 73; heterocism of, 59, 154; *P. Kuehni*, 380; *P. Lolii*, 182; *P. Malvacearum*, 161; (see *Hollyhock rust*); *P. Maydis*, 193; *P. Pennsylvæ*, 223; *P. purpurea*, 194, 206. *P. simplex*, 185; *P. triticea*, 151; sub-stomatal vesicle of, 69; *P. Violæ*, effect on flowers, 89.  
*Pyrenidium*, 19.  
*Pyrenospores*, 24.  
*Pyrenophora trichostema*, 184, 180.  
*Pythium de Baryanum*, damping off caused by, 79; does not destroy starch, 52; parasitism of, 44; penetration of cell walls by, 95; sporiferous mycelium (cf. 17; *P. gracile*, 348; *P. ultimum*, effect of nutrition on, 53.  
 Radish, diseases of, 201.  
 Rag leaf blight, 241.  
 Rai (see *Brassicæ*).  
 Rala (see *Kangni*).  
*Ramularia areola*, 308.  
 Rangoon bean (see *Bean*).  
*Ranunculus*, dormant mycelium in, 58.  
*Raphanus sativus* (see *Radish*).  
 Raspberries, *Coniothyrium* on, 119.  
 Reactionary cork, 99, 100.  
 Rennetase in fungi, 52.  
 Resin, as an adhesive in Bordeaux mixture, 142; flux in conifers, 84, 102; in fungus cells, 9.  
 Resistance to disease, 114; influence of climate on, 117; selection and breeding for, 125.  
 Retting of flax and jute, 86.  
 Reversion caused by fungus attack, 91.  
*Rhamnus*, *Puccinia* on, 93.  
*Rhizoctonia*, clamp-connections in, 9; damping off caused by, 79; hosts of, 268; sclerotia of, 12, 13; specialisation of

- parasitism in, 61, 266; stromata of, 16;  
*R. destruens*, 290, 323; may enter  
 through lenticels, 67; *R. Solani*, 262;  
*R. species* on brinjal, 305; on cotton, 370;  
 on cowpeas, 12, 262; on groundnut, 323;  
 on jute, 12, 372; on opium poppy, 346;  
 on potato, 290; on tomato, 305; on val,  
 267.
- Rhizomorphs, 10, 11.
- Rhizopus nigricans, consumes pectic bodies,  
 96.
- Rice brusone, 128.  
 bunt, 226.  
 false smut, 228.  
 sclerotial disease, 230.
- Ricinus communis (see Castor).
- Ringing wound, 82.
- Ripe rot of fruit, 83, 109, 122.
- Roselia may prevent cork-formation, 104.
- Rosa, mildew on galls of, 124.
- Rose, Corticium salmonicolor on, 499; leaf  
 disease of, 88, 95 (see Diplocarpon Rose).
- Rosellinia, most severe in acid soils, 112;  
*R. arcuata*, 437; *R. bunodes*, 357, 436;  
*R. echinata*, 436; *R. necatrix*, 358; *R.* on  
 coffee in Ceylon, 481; *R. Pepo*, 359, 436.
- Rosellinias of tea, 455.
- Rotation, advantage of, 107.
- Rots caused by fungi, 83, 95, 103, 122.
- Rottboellia, sclerotial disease of, 411.
- Rotting of fruit and potatoes, 109.
- Rubber anthracnose, 512.  
 black thread, 494; influence of  
 shade on, 117.  
 brown root disease, 506.  
 die back, 509; infection by, 67.  
*Fomes lignosus* on, 506.  
*Fomes somitostus* on, 506.  
 leaf fall, 494.  
 leaf spot, 515, 516.  
 Pestalozzia on Hevea, 455.  
 pink disease, 499; thyloses in, 102.  
 pod rot and canker, 490.  
 red root disease, 432.  
*Rosellinia* on Castilloa, 436.  
 stump rot, 506.  
*Uetulina zonata* on, 440.
- Rust, influence of humidity on, 110.
- Rusta, dormant life of, 45, 58, 160; factors  
 influencing resistance to, 121; hetero-  
 cecism of, 59; infection by, 64, 66, 69;  
 specialisation of parasitism in, 60.
- Saccharomycetes, gemmate mycelium of,  
 14.
- Saccharum officinarum (see Sugarcane).
- Saccharum spp., ring spot on, 381; rust on,  
 390; sclerotial disease on, 411; smut on,  
 380.
- Sanitation, principles of, 106.
- Sann hemp rust, 373.
- Sap-wood, action of fungi on, 97, 98.
- Sapodella, Corticium salmonicolor on, 499;  
 thread blight on, 457.
- Saprolegnia mixta, effect of nutrition on,  
 52.
- Saprophytes, 41.
- Saxon (see Brassicae).
- Sawan smut, 238.
- Scab, 81, 100.
- Schizophyllum commune, sporophores of,  
 18.
- Schleichera, Rosellinia bunodes on, 359.
- Soila smut, dormant mycelium of, 58.
- Sclerospora graminicola, 218, 232; myce-  
 lium in tissues, 94; haustoria of, 50;  
 leaf-like stamen formed through action  
 of, 89; phyllody caused by, 89, 104,  
 proliferation caused by, 89; specialisa-  
 tion of parasitism in, 61, 223; *S. gramin-  
 icola* var. *Andropogonis-Sorghis*, 203;  
*S. macrospora*, 223; *S. Maydis*, 191.
- Sclerotia, 12, 58.
- Sclerotinia, appressoria of, 70; may infect  
 through flower, 68; dissemination by  
 insects, 35; *S. cinerea*, resistance of  
 plums to, 119; *S. heteroica*, heteroecism  
 of, 59; *S. Libertiana*, influence of phos-  
 phate on, 112, 123; *S. Trifoliorum*, infec-  
 tion by, 70.
- Sclerotium on sugarcane, 408; *S. Oryzae*,  
 13, 230, 410; ohlmydespores of, 14;  
*S. rhizodes*, action of, 52; *S. stipitatum*,  
 12, 13.
- Scolecospores, 391.
- Seed, diseases carried by, 109; growth of  
 resistant potatoes and sugarcane from,  
 126, 127.
- Seed disinfection, 135.
- Selection, and disease, 126; of seed and  
 cuttings, 109.
- Septa, 6.
- Septoglossum Arachidis, 319; *S. Manihotis*  
 310; *S. Mori*, stroma of, 15.
- Septoria gramineum, 175; *S. maculosa*,  
 492; *S. piriicola* attacks chlorotic plants  
 most readily, 124; *S. Tritici*, 175.
- Sesamum, Bacillus solanacearum on, 338;  
 Phytophthora on, 330; Rhizoctonia on,  
 263.

- Setae*, 218.  
*Setaria italica* (see Kangni).  
*Setaria viridis*, smut of, 303.  
 Sexual reproduction, 20, 25.  
 Shade, influence on asparagus rust, 111;  
     on black thread of rubber, 498; on blister  
     blight of tea, 429; on celery leaf spot, 111,  
     316; on chilli die back, 111, 354; on  
     Colocasia blight, 308; on copper blight  
     of tea, 444; on cucumber mildew, 111;  
     on infection, 72; on pink disease of rubber,  
     503; on strawberry leaf disease, 111; on  
     thread blight of tea, 480; on tobacco  
     mildew, 111, 339; on wheat mildew, 111,  
     175; physiological effect of, 117, 120.  
 Shade trees, and *Botryodiplodia Theobromae*,  
     445; and brown root disease, 430, 479;  
     and red rust, 421; and sooty  
     mould, 480; and stump rot, 358, 442,  
     480.  
*Shorea robusta*, action of *Polyporus*  
*Shorea* on, 98; "horse hair" fungus on,  
     11.  
 Shot hole, 81, 100.  
*Sicyos angulatus*, mildew of, 124.  
*Sida cordifolia*, *Phytophthora* on, 330.  
*Sisal anthracnose*, 374.  
 Smoke, injurious effect of, 116.  
 Soil, disinfection, 135; influence on *Botryo-*  
*diplodia Theobromae*, 447; on cotton of  
     alkali, 370, 372; on disease, 117, 124,  
     119, 474; on early blight of potatoes,  
     289; on grey blight of tea, 454; on  
     *Hymenochaete noxia*, 431; on red root  
     disease, 434; on red rust, 421, 422; on  
     *Rosellinia*, 442; on soft rot of ginger,  
     351; on sugarcane red rot, 401.  
*Solanum Commersoni*, *Alternaria Solani*  
     on, 290.  
*Solanum Molongena* (see Brinjal).  
*Solanum nigrum*, *Alternaria Solani* on, 290.  
*Solanum tuberosum* (see Potato).  
 Sooty moulds, polymorphism in, 30.  
*Sorghum* (see Jowar).  
*Sori*, 18.  
*Sorosporium Paspali*, 240.  
 Soy bean downy mildew, 266.  
 Specialisation of parasitism, 60, 114.  
*Spermagonium* of rusts, 29.  
*Spermatia* of rusts, 29.  
*Sphaerotheca oruenta*, 212; *S. Sorghi*, 208.  
*Sphaerella Camelliae*, 443; (see *Mycos-*  
     *sphaerella*).  
*Sphaeronema adiposum*, 382.  
*Sphaerostilbe repens*, 432, 506.  
*Sphaerotheca Humuli* var. *fuliginea*, 314;  
     *S. pannosa* on peach and almond, 45;  
     *S. phytophila*, localised infection  
     by, 68.  
*Spinach* downy mildew, 318.  
*Spondylocadium*, potato scab caused by,  
     81.  
*Spongospora*, potato scab caused by, 81.  
*Sporangium*, 21.  
*Spore*, types of, 24.  
*Spores*, dissemination, 32; formation, 20;  
     germination, 6, 70; numbers, 32; pro-  
     perties, 32; "puffing", 33.  
*Sporidia*, 24, 29.  
*Sporodesmium Brassicae*, 301.  
*Sporodochia*, 354.  
*Sporophores*, 16.  
*Sprayers*, 133.  
*Spruce*, *Polyporus subacidus* on, 98; *Trametes*  
*Pini* on, 96.  
 Stag headed condition of trees, 79.  
 Starch, action of *Fusarium* on potato, 93;  
     changed to sugar by fungi, 51; consumed  
     by *Botryodiplodia Theobromae*, 510;  
     consumed by some fungi, not by others,  
     52, 93; increased by some fungi, 93.  
*Stereum frustulosum*, action on wood, 98;  
     *S. hirsutum*, attacks sap-wood, 97; *S.*  
     *subpileatum*, attacks heart-wood, 97.  
*Sterigmata*, 24.  
*Sterigmatocystis*, effect of nutrition on, 53.  
*Stigmatae Robertiana*, sub-cuticular growth  
     of, 99.  
*Stilbum nanum*, 480.  
*Stizolobium* (see Velvet bean).  
 Strawberry diseases, influence of shade on,  
     111.  
*Stromata*, 15.  
 Stump rot, 357, 435, 479, 506.  
*Stylospores*, 24.  
 Sub-cuticular parasites, 99.  
 Sub-stomatal vesicle, 66, 69.  
*Suberin*, 99.  
 Sugar, inverted by some fungi, 51; in  
     fungus cells, 8.  
 Sugarcane, deterioration of, 127; insects  
     may lower resistance of, 116, 401.  
 Sugarcane banded sclerotial disease, 408.  
     black rot, 382.  
     brown leaf spot, 404.  
     *Cercospora vaginæ* on, 70.  
     collar rot, 388.  
     dry rot, 385.  
     helminthosporiose, 406.  
     pine-apple disease, 385.

- red rot, 116, 117, 122, 126, 128, 391.  
 ring spot, 381.  
 rust, 380.  
 smut, 377.  
 sooty mould, 411.  
 wilt, 402.
- Sulphur as a fungicide, 131, 144, 146.  
 Sulphur dusting, beneficial effects of, 138.  
 Sycamore, *Polyporus squamosus* on, 97.  
 Symbiosis, 53.  
 Symmetry altered by fungus attack, 90.  
 Synohytrium, hypertrophy caused by, 85, 104; *S. decipiens*, effect on nucleus, 93, 94; *S. mercurialis*, hypertrophy caused by, 104; *S. Pueraria*, effect on cell contents, 93; *S. pyriforme*, effect on cell contents, 93; *S. trichophilum*, localised infection by, 68; *S. viride*, effect on chlorophyll, 93.
- Tannin consumed by some fungi, 52; toxic to some fungi, 94, 122.  
*Taphrina Laurencia*, 89; *T. maculans*, 346; mycelium and haustoria of, 48; *T. rhomboidalis*, mycelium and asci of, 48, 49.  
*Tapia*, *Botryodiplodia Theobromae* on, 386; (see *Cassava*).  
 Tar, protection of wounds by, 108, 498, 504, 505, 512.  
 Tea, disease resistance of varieties of, 422, 429; influence of vigour of bush on diseases of, 73, 115.  
 Tea black blight, 463.  
   blister blight, 422.  
   brown blight, 448.  
   brown root disease, 429.  
   canker, 83, 463.  
   *Capnodium Footii* on, 462.  
   copper blight, 443.  
   *Corticium Theae* on, 460.  
   *Crepidotus* on, 460.  
   die back, 450; influence of nitrate on, 123.  
   grey blight, 73, 413, 461.  
   "H" blight, 450.  
   *Hendersonia theicola* on, 451.  
   internal root disease, 445.  
   leaf spot, 455.  
   *Nectria cinnabarina* on, 463.  
   *Penicillium* in seed of, 465.  
   pink disease, 429.  
   *Poria lateritia* on, 435.  
   red root disease, 432.  
   red rust, 413.  
   rim blight, 455.  
   seed mould, 465.  
   seedling disease, 464.  
   sooty mould, 461.  
   stump rot, 435.  
   thread blight, 47, 456.
- Teak, *Corticium salmonicolor* on, 409.  
 Teleutospores, 24, 29.  
 Temperature, influence on germination of *Hemileia* spores, 472; on potato blight, 282, 293; on spores of *Cystopus candidus*, 71, 296; on spore germination, 71, 110.  
 Thickness of cell walls influences penetration, 68, 72.  
*Thielaviopsis*, on palms, 385; *T. paradoxa*, 385.  
*Thujopsis*, *Caeoma* on, 89.  
 Thyloses, 102.  
*Thyridaria tarda*, 385, 445, 509; parasitism of, 44.  
 Til leaf disease, 330; (see *Sesamum*).  
*Tillotia horrida*, 226; *T. levis*, 166; *T. Oryzae*, 228; *T. Tritici*, 166.  
 Tobacco, *Bacillus solanacearum*, causes gum formation in, 101, 102; causes leaf galls on, 89.  
 Tobacco bacterial wilt, 332.  
   leaf spot, 340.  
   mildew, 338.  
   mosaic disease, 341.  
   *Pythium gracle* on, 352.  
   *Rhizoctonia* on, 263.  
*Tolyposporium filiforme*, 215; *T. Penicillariae*, 224.  
 Tomato, influence of fertilisers on disease of, 124; intumescences formed by *Bacillus solanacearum* on, 89.  
 Tomato bacterial wilt, 303.  
   blight, 304.  
   fruit rots, 304.  
   mosaic disease, 342.  
   *Phytophthora Faberi* on, 490.  
   *Phytophthora parasitica* on, 330.  
   root rot, 305.  
   wilt, 304.
- Toria (see *Brassicaceae*).  
 Torula stage of sooty moulds, 30.  
 Toxic action of fungi, 51.  
 Toxin production by *Bacillus solanacearum*, 335.  
*Trachoscleria*, *Botrytis cinerea* on, 102.  
*Trametes Pini*, alterations in wood caused by, 83-96, 97, 98; tannin as food for, 52.  
*Tremellinaceae*, gelatinisation of wall in, 10.

Trehwella Jack for uprooting trees, 481.  
 Trichogyne, 26.  
 Trichosanthos (*see* Cucurbit).  
 Trichosporium vesiculosum, 86.  
 Trichothecium candidum, 43.  
 Trifolium, Erysiphe Polygoni on, 61.  
 Trigonella Foenum-græcum (*see* Fenugreek).  
 Triposporium stage of sooty moulds, 30.  
 Triticum (*see* Wheat).  
 Tumours, 84; on coriander, 339.  
 Turmeric leaf spot, 346  
 Turnip (*see* Brassicas).  
 Turnips, "finger and toe" in, 103, 107.  
 Tyrosinase in fungi, 52.

**Uredo** echinatus, appressoria of, 69; U Gossypii, 363; U. operta, 242; U Paspali-scribiculari, 240; U. species on Panicum miliaceum, 238.

Uredospores, 24, 29; infect and develop best on healthy plants, 116

Urid (*see* Bean).

Uromyces occulta, 173; U Tritici, 171.

Uromyces Anthyllis, 362; U. appendiculatus, 260, 262, 267; U Betæ, 123; U. Cicoria-arietini, 271; U decoratus, 373; U. Fabe, 29, 254, 268; polymorphism in, 29; specialisation of parasitism in, 255; U Hobsoni, hypertrophy caused by, 103; U. Kuehnii, 380; U. linearis, 238; U. Mucunæ, 268; U. Pisi, 256; effect on host of, 91; U. Setariae-italicæ, 233; U. species on Khosari, 268; U. Trigonellæ, 362.

Ustilaginoides virens, 228.

Ustilago Avenæ, 179; U. Coicis, 242; U. Crameri, 234; U. emodensis, gall formed by, 89, 104; U. Hordei, 184; U. nuda, 184, 185; U. Panicifruentacci, 238, 239; U. Panicimiliacei, 235; U. paradoxæ, 238, 239; U. pulveracea, 199; U. Reihana, 199, 214; U. Sacchari, 377; U. Tritici, 163; U. violaceæ, 61, 88; U. virens, 228; U. Zææ, 194; gummy degeneration caused by, 101; infection by sporidia of, 66; invert cane sugar, 51; prevents outinisation of outer wall, 102; prevents lignification of vessels, 102; proliferation caused by, 89; spores of, 21.

Ustilula vulgaris, 440; U. zonata, 435, 438, 506.

Vaccinium, Sclerotinia on, 60.

Vacuoles, 7.

Val anthracnose, 267.

die back, 267.

root rot, 267.

rust, 267.

Valsa oxystoma, influence of soil on, 117.

Vanillin, 96.

Vegetable marrow, influence of shade on mildew of, 111; (*see* Cucurbit).

Vegetative mycelium, 6.

Velvet bean rust, 268.

Venturia, appressoria of, 70; effect of outicle, on infection by, 72; germination of spores of, 70; growth on resistant hosts, 121; scab caused by, 81; sub-cuticular growth of, 99; V. circinans, consumes cellulose 95, 99; poetic degeneration caused by, 102; V. inæqualis, apple scab caused by, 82; division of cells caused by, 101; effect on chlorophyll, 93; formation of reactionary cork by, 99; hypertrophy caused by, 101, 104; infection, 72, 99, 121; may increase calcium oxalate, 93; may leave starch unaltered, 93; penetration of pear by, 121; (*see* Apple scab); V. pirina, influence of shade on, 72; penetration of apple by, 121; penetration of reactionary cork by, 99; (*see* Pear scab).

Vermicularia Capsici, 262, 267, 304, 305, 352; V. sp from Carica, nuclei of, 9.

Vicia, dormant mycelium in, 58; V. Faba rust, 254.

Vigna Caljang (*see* Cowpea).

Vine black rot, influence of age of host, 72; of humidity, 117; of reaction, 112, 121; of weather, 444; (*see* Guignardia Bidwellii).

Botrytis, 72.

grey rot, influence of nitrate, 122.

mildew, influence of shade, 111; perithecia, 58; rate of spore-fall, 70; resistance to, 114, 122.

Phylloxera, 127.

white rot, resistance of waxy varieties; 72.

Viola, Puccinia on, 89.

Virulence, 112; loss of, in Alternaria Solani, 289; variation in, 43, 112.

Watermelon, wilt-resistance in, 118, 126.

Waxy coating, influence on penetration, 72, 119.

Weak parasites, 42.

Wheat bunt, 166; longevity of spores of, 32; perpetuation of, 57.



- flag smut, 171.  
 leaf spot, 175.  
 loose smut, 118, 119, 163; perpetuation of, 57.  
*Melanospora damnos* on, 124.  
 mildew, 62, 124, 173; influence of fertilisers, 123; of shade, 218, 111, 175; perpetuation of, 58.  
 mould, 177.  
*Rhizoctonia destruens* on, 291.  
 rust, 59, 114, 118, 126, 151; dormant mycelium of, 45; factors that cause epidemics of, 129, 162; influence of fertilisers, 122, 123; of humidity, 110, 162; of reaction, 121; of shade, 124; perpetuation of, 159; specialisation of parasitism in, 61, 63, 154, 186.  
 White-lead paint for wounds, 108.  
 Wilt disease, 44, 75, 79, 244, 251; influence of air on, 112.  
 Wind breaks, 109.  
 Witches' brooms, 86, 87, 91, 92, 103; perennial mycelium of, 58.  
 Withering, 78.  
 Wood gum, action of fungi on, 95, 97.  
 Wound gum formed by action of fungi, 102  
 Wound parasites, 44, 67.  
 Wounds, caused by fungi, 82; influence on susceptibility, 63, 125; treatment of, 108, 493, 504, 512.  
 Xylan consumed by some fungi, 95.  
*Xylaria nigripes*, *sclerotium* of, 13.  
 Yeasts, 14.  
*Zea Mays* (*see* Maize).  
*Zingiber officinale* (*see* Ginger).  
*Zingiber* spp., *TaPhrina* on, 347.  
 Zygospor, 25.